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SUGGESTIVE REPERSONALIZATION

THE PSYCHOPHYSIOLOGY OF HYPNOTISM *

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I. WHAT IS HYPNOTISM?

In presenting this topic of hypnotism for discussion, I have thought it opportune to reconsider the subject, notwithstanding all that has been written about it, for in my judgment more superficial, unscientific observations have been made, more specifically selected data to bolster up particular hypotheses have been collected, more ill considered conclusions have been drawn, and more nonsense has been written than in any other important problem. The terms hypnotism and hypnosis have acquired unfortunate implications and connotations and are not free from stigma and prejudice even in the minds of physicians. In the minds of lay people, they border on the occult. Furthermore, they do not correctly define the phenomena. Some new and more correct term is therefore desirable—one that will represent the psychophysiologic principles involved and will be descriptive of the phenomena.

For there is a general misunderstanding of the nature of hypnosis, leading to many foolish questions, such as: "How many persons can be hypnotized?" This and many other questions indicate a misunderstanding of the nature of hypnosis. It is equally correct to answer: "Very few can be hypnotized," or "Every one can be hypnotized," according to the understanding of hypnosis.

However, this is of small importance. My main thesis is that hypnotism has always been treated as if it were something bizarre, a mental condition that stood apart as something distinctly different from all other conditions; whereas it is only one of a large category of conditions characterized by alteration of the personality. In this category are to be found various clinical types of alteration, some normal and some abnormal, all due to the same processes and mechanisms, and therefore fundamentally resembling one another, in that they are all types of depersonalization and repersonalization from the standpoint of

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the modern conception of the structure of personality. Specifically, these types are known as attention, abstraction, revery, sleep, hypnosis, moods, ecstasy, trance, fugues, somnambulisms, mutiple personality, etc. The only reason for differentiating hypnosis is that it is an artefact most commonly induced by a specific external stimulus (suggestion); on the other hand, it is not always so induced, but may be induced by endopsychic stimuli (autosuggestion), while, correspondingly, the other types may also be induced as artefacts by external suggestion, though they are more commonly induced by endopsychic stimuli.

A popular conception, shared by many medical men, is that there is some definite specific state into which a personality becomes transformed when hypnotized. Of course there is no specific definite state constituting hypnosis, any more than there is any specific waking state, in which alertness or torpidity, absentmindedness, abstraction, fantasy or daydreaming, intoxication, moods with alterations of character traits, acute emotional states, etc., are present. These states all vary in their psychologic and physiologic traits. Consequently, the states of hypnosis are as varied and multiform as there are possible combinations of the psychologic and physiologic components of personality. Even in the same person several different states may develop, each exhibiting different memories, traits, and other personality characteristics, and physiologic aberrations, such as anesthesia and paralysis.

The recognition of this fact is important practically, because when hypnosis is employed for experimental research, psychologic analysis, or therapeutics, an examination of the reports often reveals that it has been assumed by the author as a matter of course that the succeeding hypnotic states, induced in the course of successive observations in the same subject or patient, are always the same as that first obtained; whereas it is the case, as a careful reading of such a report makes clear, that a different state, or integrate, was unwittingly obtained exhibiting different memories, traits and other phenomena. This error may vitiate the conclusions.

However varied and multiform the states of hypnosis, there are certain general principles to which every state conforms. These are inhibition, dissociation, and synthesis or reintegration. It is generally agreed that every hypnotic state involves inhibition and dissociation, but it is not so generally realized, and it is certainly not emphasized, that hypnosis also involves reintegration, often as the preponderant alteration, with a total result of depersonalization and repersonalization.

This is true because the induction of this state is and must be governed by the normal mechanisms of the mind, for every act of attention, thinking, reasoning and perception, however motivated, involves inhibition, dissociation and integration, just as on the physiologic side, as Sherrington has shown, the same holds true for the

sensorimotor activities of the nervous system and must be true of the brain as well as of the spinal cord. All conflicting mental systems of activity mutually tend to inhibit one another, so that when one system, say, of thought or perception, is in activity, it tends to inhibit other systems that would conflict or would be incompatible with it if in activity. This inhibition is particularly intense when the inhibiting system is motivated by an emotion. Unless this were true, the functioning of the mind and nervous system would be chaotic. The simple normal states, for example, of active and passive abstraction, attention, contemplation, revery, "brown-study" and absentmindedness, are the product of these three processes; abstraction is, as I will later point out, identical in principle with light hypnosis.

In other words, light hypnosis, the stage to which the great majority of subjects are hypnotized, is nothing more or less than a particular type of abstraction.

PHENOMENA

Let us take the two extreme types of hypnosis, the so-called lighter and deeper states (mere figures of speech, by the way), and tabulate the phenomena as determined by observation. Between the two extreme types, every degree and variety of dissociation, inhibition and integration occur. Dissociation and inhibition with resulting depersonalization may predominate, or it may be integration; or both may be in equal proportions. The final repersonalization may have for an end product such an extreme alteration of personality as to result in a complete change of character and be identical with what is known as a secondary personality.

It will be well to examine first the extreme type, so-called somnambulism, for in this type the phenomena resulting from the three processes are so obtrusive that they cannot escape recognition.

DISSOCIATION AND INHIBITION

It is advisable to consider dissociation and inhibition together, for though they are not coextensive or identical as phenomena, nevertheless in practice it is often difficult to distinguish between them, for they are undoubtedly only different results of the same process. Though the terms are commonly used synonymously, this use is not strictly correct. Dissociation is the more generic term, for, while that which is inhibited is also dissociated, that which is dissociated is not necessarily inhibited. A sensation, for example, may be only split off from the content of the consciousness of the moment, as perceptions in the fringe of the conscious field are split off from the attentive consciousness (i. e., the focus of attention). In this split-off state the sensation may function involuntarily or automatically, as it is called, or it may be capable of so functioning, and then is not inhibited but only dissociated from the field of awareness.

Mental Dissociations.—On the mental side, dissociation (and inhibition) may involve the memories of whole epochs in the subject's life, so that he reverts to a period long antedating the day of hypnosis. There is, therefore, complete amnesia for the succeeding periods, and he lives again in a preceding epoch. Thus, Mrs. J., when hypnosis is induced, reverts suddenly and unexpectedly back nine years to the day just before she received an emotional shock, and imagines it is that day. There is complete amnesia for the whole intervening time of nine years. Similarly, Mrs. B., a widow, reverts two or three years to a period when her husband was alive and under that illusion mistakes me, to my embarrassment, for her husband and proceeds to enter into confidences of family life of a specific day in the past.

Again the dissociation may involve intellectual acquisitions such as languages, and it is well to bear in mind that different states mean different dissociations; thus, Miss B., at one time in hypnosis has complete amnesia for Latin and at another for French, although she knows both well.

Some of the most striking alterations of personality (depersonalization) result from the dissociation of traits characterized by sentiments, deeply cherished beliefs and ideals. Thus, intensely held religious sentiments and beliefs may be completely dissociated and lost in one or another phase. Likewise, sentiments of aversion or affection toward persons may disappear and may be replaced by their opposites. This substitution may be traced to a reversion to the sentiments that obtained toward the same object at an earlier epoch of life. B. C. A. acquired in an *A* phase a most intense aversion amounting to scorn and hatred toward a certain person X with whom, at an earlier epoch, she had held pleasant and friendly relations; in the *B* phase, the earlier sentiment of friendship recurred and became reintegrated with the personality of this phase.

The parental sentiment may likewise be completely dissociated and lost, only to be immediately recovered on emergence from the hypnotic phase. In consequence of this dissociation, a repersonalized subject took not the slightest interest in her young son, who she felt, as she said, was not her son, although she was conscious of the fact that he was born of her body. Do not imagine that this was a phenomenon of suggestion. On the contrary, it occurred spontaneously as the result of psychic conflict.

Equally instructive is the disappearance through dissociation of the self-regarding sentiment and its replacement through reintegration of its opposite. I could cite several examples of this. One will suffice. Mrs. O. has a conception of self characterized by a feeling of inferiority, incapacity and weakness of will. In the hypnotic phase, this is replaced through reintegration by a sentiment of self-pride, superiority, dominating capacity and will, self-assertion, superior intelligence, etc.

A curious and psychologically interesting phenomenon that I observed in one case was the dissociation in one phase of hypnosis, known as "alpha and omega," of the conception or idea of self. This phase described her consciousness as only "thought" without any conception or feeling or idea of being an ego or I—nothing to which the word "I" could be applied—just thought without an I.

Equally curious was the loss of the time sense that "Sally" exhibited. One minute, or an hour, or a day, was the same to her.

A phenomenon of dissociation effecting a marked alteration of personality is difficult to formulate. It involves a psychologic principle for which I have contended for a long time. This dissociation is of a large system of mental dispositions which, with their roots, acquired by experience, gives as the setting that particular "meaning" to objects and ideas that they have for the individual and determines the attitude of mind and point of view of the individual toward such objects and ideas. A good example would be the difference in the meaning of "mother" to you and to me, according to whether it is your mother or my mother. The difference, of course, depends on the difference in our individual experiences. In consequence of the dissociation of such a system, particular meanings of persons, places, objects, situations, etc., and emotional attitudes toward them are completely lost and perhaps replaced by others belonging to an earlier epoch of life. The latter are then reintegrated with the hypnotic personality. For example, an outdoor life, the woods and canoeing, are objects of intense distaste to Mrs. B. and are avoided so far as possible; in the *B* phase an absolutely opposite meaning and point of view with corresponding tastes and behavior emerge, and determine her choice of activities. In my view this principle is of the utmost practical importance and is the basis of psychologic therapeutics. I could cite example after example of this principle if time permitted.

In the affective field, dissociation and depersonalization may be equally distinctive. Here one may have to do with primitive unintegrated emotional instincts and appetites. This form of depersonalization has for the most part been overlooked, although, I may say, I have frequently described it as of great importance in my studies of dissociated personality. I will content myself with a mere enumeration of some of the dissociated or inhibited instincts. Fear was totally absent in more than one case; the loss of hunger was noted in another. The sex appetite or instinct may likewise be totally inhibited in some one hypnotic phase of personality and may be present in another phase. Thus in one phase of hypnosis, Mrs. O. was totally asexual, and in another, intensely sexual. The same loss of the instinct occurred in one phase with Mrs. B., in which phase the term had lost all but a dictionary meaning.

Similarly, anger and the other emotional instincts may be dissociated with corresponding depersonalization. I will further cite in this category only the complete loss of the instinct of self-assertion with the consequent dominance of self-abasement in one hypnotic phase of Mrs. O., while in another phase the reverse was the case, and the subject exhibited the most exalted self-assertion and domination of her environment.

All these mental dissociations occurred spontaneously, independently of suggestions, and characterized the hypnotic phase.

Physiologic Dissociations.—On the physiologic side the personality may lose spontaneously a variety of functions, the most conspicuous being those of sensation and motility. Thus, B. C. A. could be put into a number of different hypnotic states. In one, there was not only general loss of tactile sensibility over the whole body, but also complete loss of cenesthesia. She had no consciousness of having a body at all, no awareness of muscular movements, including the mimetic movement of the facial muscles. She did not know whether she was standing or sitting. She described herself as "thought in space." In another state there was loss of the special senses of taste and smell. In the state I call alpha and omega, tactile sensibility was lost.

The sensory functions dissociated in one hypnotic state were reintegrated in another. Likewise, spontaneous paralyses have been noted, although I have not observed them. As with the mental dissociation, these physiologic forms occurred spontaneously.

INTEGRATION

I now come to integration and repersonalization. The most familiar phenomenon is the integration in hypnosis of dispositions pertaining to memories of past experiences, forgotten and beyond recall in the waking state. These may include experiences of another state for which there is complete amnesia in the waking state. Among the latter are those of sleep (dreams), trance, fugues, intoxication, delirium, nocturnal somnambulism, amnesic states, and the experiences of a second, third, or fourth dissociated personality. These are all phenomena of integration and are components of a new integrate.

Less familiar is the integration and recall or memories of perceptions of the environment that never entered consciousness at all but were only subconsciously experienced, or at most were in the fringe of consciousness. I have made various experiments demonstrating this phenomenon. It should also be recognized that such perceptions may be integrated in one hypnotic state (*a*) but not in others (*b* and *c*).

Sometimes such subconscious perceptions become integrated in dreams; or they may become manifested in strikingly dramatic form as visions simulating "thought transference" or spiritualistic phenomena, as I have on several occasions observed. Thus, for instance, a telephone message received by my secretary from a distance and transmitted as a cablegram to the cable company was read in a crystal by a subject of mine. She had subconsciously heard the message as it was transmitted, without being consciously aware of the fact.

Repersonalization through the process of integration is particularly striking when large systems of memories, thoughts, reflections and fabrications in the form of fantasies become so integrated with the personality as to stamp the make-up with a character of dramatic individuality. Mrs. O., for instance, in a dissociated state known as Maria, manifested a dominating fantasy of being the reincarnated soul of a Spanish peasant girl and (later) a courtesan of the thirteenth century. She spoke broken English and a supposed Spanish dialect (a neologism, of course) and sang, danced and acted the part in a most characteristic and entertaining way. In other respects, she appeared to have a normal personality.

The fantasy was the reintegration of youthful day dreams which had undergone subconscious incubation and had grown until finally they burst into flower. This fantasy was thoroughly believed in, became the dominating trait of the personality combined with a number of other reintegrated sentiments, instincts and traits. They were components of a large, highly organized integrate, the psychogenesis of which I was able successfully to unravel. The case was one of complete, thoroughgoing repersonalization.

Of the common traits that make up personality, the reintegration of sentiments is the most instructive from this point of view; but I have already, when discussing the phenomenon of dissociation, cited examples enough of the reversion to sentiments entertained at an earlier period of life and the substitution of an earlier and opposite sentiment for a later one regarding one and the same object. The alterations of personality occasioned by such integration may be readily recognized. I have likewise spoken of the substitution of earlier meanings of objects, ideas and situations, and of corresponding attitudes of mind and points of view. The repersonalization affected by such reintegrations must be obvious.

I have already said enough, too, about the emotional instincts as such. I will merely add that I have more than once observed the reintegration of the sex instinct and some others (anger and self-assertion) that were repressed and inhibited in the normal waking state.

Moods.—The depression and exaltation of a waking state may be spontaneously replaced by their opposites. Depression gives place to

exaltation, and vice versa. Of course a mood involves, besides the affects peculiar to it, large systems of mental dispositions determining the point of view, attitudes of mind toward and meaning of the situations of life. I cannot enter into this here. However, it is easy to understand how the integration of one system with its affects in place of another may effectively contribute to the repersonalization of the individual.

In illustration I might cite many experiments and observations of my own, but to eliminate personal bias, I shall mention an interesting experiment by another observer.

While engaged in some hypnotic experiments on a man, aged 22, who was "essentially normal and responsible, of robust character and of decided intellectual ability," "Greenwood"[1] found to his surprise that suddenly, entirely independently of suggestion and as if by accident, the young man fell into at least four distinct phases or moods, each of which may be well characterized as a self.

The first phase, the ordinary or quiet mood, was similar to his normal self when awake. He was of a quiet nature, speculative and restrained, well bred and courteous in demeanor and of a religious and idealistic temperament. If a suggestion was made not consonant with this character, it was rejected at once and any amount of insistence would be in vain.

In the second phase, called the "gay mood," into which, on its first appearance, he suddenly, *without warning* and to the surprise of the experimenter, changed out of the first phase, the subject became extremely hilarious and absurd, jested in an easy way, displayed a tendency to practical jokes on the experimenter, kicked his clothes about the room and was generally obstreperous and fantastic, both in speech and in behavior. Then, of a sudden, without warning or suggestion of any kind, he reverted to his former quiet, gentle, restrained self. On other occasions in this gay mood, which frequently occurred, he showed himself to be a "gay Lothario," for he displayed an astounding lack of the ordinary conventions or proprieties, professed a complete contempt for either religion or morality and a disregard for any responsibility in his actions, becoming in his own language, a child of nature, nonmoral though not vicious. Any suggestion not consonant with this mood was, as in the first phase, instantly rejected.

The third phase was a "malicious" mood. In this he became a sort of "Jack-the-Ripper." He exhibited a strong wish to inflict pain and frequently asked permission to stab the experimenter in order to have the gratification of seeing blood flow. Indeed, he was detected surreptitiously extracting a penknife from his pocket with a view to satisfying this inclination. He confessed to a wish to vivisect, or, failing that, to strangle.

The fourth phase into which the young man fell in the same way was a "depressed" mood, the very opposite of the gay phase. Now he exhibited himself as a melancholic—a melancholy Jaques—utterly and beyond bounds miserable and ready, for no reason that was apparent, to burst into tears.

Each of these moods, or so-called selves, carried its own different set of emotions, tastes and mental attitudes. As I have said, suggestions not consonant with the particular mood he was in were rejected. The whole manner of the man in each exhibited an absolute contrast of expression, conduct and mode of speech, just as we all do in our different moods, but not like those of this young man, I hope.

1. "Greenwood, Edward:" Society for Psychical Research, Proceedings **17:** 279, 1903.

As to the reintegration of physiologic functions, sensibility and motility that have been inhibited in the waking state, manifested by functional anesthesia and paralyses, may be reintegrated in the hypnotic state. As evidence of the integrating effect as contrasted with the dissociating effect of the hypnotizing procedure, nothing is more instructive and convincing than the results of a certain technic I have successfully used in restoring the complete normal personality out of two different phases of multiple personality. The technic has been to hypnotize one or both of the different dissociated personalities, *A* and *B*, into deeper and deeper states until one, *C*, is obtained that is a completely integrated composite of the two. Each is thus reintegrated in this one *C*, which is found to be the whole normal personality. The technic is precisely the same as that of conventional hypnotizing. The suggestions have been precisely the same; namely, "sleep," or "sleep deeper" and yet the process was almost entirely that of integration. The combined memories of *A* and *B* were integrated in *C;* the instincts and other traits dissociated respectively in *A* and *B* were restored and so with the other components of personality. Likewise, I may cite a classic case, one of Jules Janet's, that was supposed to be the normal personality but was a dissociated hysteric state with various stigmas. When the patient was hypnotized, the stigmas disappeared, and he became perfectly normal and lived a normal life in this state. It was thought to be a hypnotic state, but far from that it was the normal personality that had been "waked up" unwittingly by the hypnotizing procedure. The previous hysteric state was the dissociated personality which now by the hypnotic procedure had become reintegrated into a normal one.

Passing in review all the phenomena I have cited as observed in so-called "deep hypnosis," I think there can be no question that they were the product of the three processes I have dwelt on and that the results obtained may be properly characterized as suggestive "depersonalization" and "repersonalization."

It will be objected that some of these examples are cases of so-called multiple personality. Of course some are, but what of it? Every one who has made intensive studies of dissociated personalities will agree that they are identically the same as the somnambulistic or the deepest stages of hypnosis as produced by hypnotic suggestion. This is well exemplified by Greenwood's case that I have quoted. In the second place, in some cases the secondary personalities were first obtained by hypnotizing as hypnotic states, as happened in three cases I have cited; it was only later that they broke loose and emerged as full fledged secondary personalities. In the third place, one finds that as the result of autosuggestion similar repersonalization frequently occurs, as exemplified by the trance states of mediums, which are generally accepted as

identical with hypnotic states, on the one hand, and with secondary personalities on the other.

Moreover, in light hypnosis the same phenomena of dissociation, inhibition and integration are found, differing only in degree and in their combinations. Between the lightest states and the more extreme repersonalizations of somnambulisms, one finds among the dissociations nearly every variety of mental and physiologic component of the mind-body (personality). And the same is true of integrations.

LIGHT HYPNOSIS

I shall now consider the phenomena of light hypnosis.

Mental Phenomena.—By the force of suggestion (whatever that may be), the attention is focused on a limited number of objects—the experimenter and his suggestions, bodily sensations and feelings of sleep. These are integrated into the content of attentive interest. The composition of this "integrate" varies largely in accordance with the suggestions of the experimenter and the expectations of the subject. It is thus an artefact. As in abstraction, it may or may not include perceptions of the environment and associated memories, which are either impossible of voluntary recall or not ordinarily recalled by the subject. It may be such a limited system of conscious processes as to constitute what has been called mono-ideism. Indeed, there may be a complete "blocking" or inhibition of "thought." The subject cannot "think."

When the integrate does not include perceptions of the environment (auditory and tactile), these may still function and may be only dissociated without being inhibited, for they may be recorded. This is shown by recovering memories of such perceptions in another state of hypnosis. Here, it should be noted, is found a similarity between light hypnosis and abstraction, revery, absentmindedness and analogous states, for perceptions of which the subject is not aware in these states have been shown in numerous observations to occur subconsciously. This is done by recovering memories of them in hypnosis and by other technical methods.

In light hypnosis, by the law of inhibition and dissociation, mental and physiologic dispositions are prevented from functioning. Incompatible ideas, the emotional instincts (fear, anger, sex, etc.) and the sentiments are in abeyance. Thus, on the one hand, critical thoughts, reflections on the suggestions given, reasoning, perceptions and images are inhibited and do not enter the content of consciousness of the moment; on the other hand, various traits peculiar to the individual, such as the instinctive emotional reactions and sentiments, for the same reason are not in functional activity and do not give a response. Thus shorn of many of its traits and reactions, the personality is more or less depersonalized.

In this way the composition of the integrate, or content of attention, is limited and may be held more or less stable by the continuing motivating force of the suggestion. But the composition, being an artefact, depends in part on the integration effected by the suggestion, wittingly or unwittingly given, and the expectancy of the subject, and in part on the field of dissociation and inhibition similarly induced. In the integrate of this light state, then, there is a slight or limited but still recognizable repersonalization of the subject.

Physiologic Phenomena.—There is an inhibition of the musculature of the body in accordance with which the muscles fall into a state of relaxation as in sleep, or in the first stages of sleep—when one is "going to sleep." This is particularly true of the elevators of the eyelids, which tend to close. Whether this relaxation is brought about by specific external suggestion or autosuggestion, by the direct volition of the subject, or by other inhibiting agencies, it is not necessary to inquire. At any rate, it is not an essential phenomenon of hypnosis.

I have said that the field of the content of awareness in light hypnosis is limited to a few specific objects such as I have mentioned and is apt to be stable, but this is not necessarily so. If express directions are given to the subject, a succession of associated memories will stream through the mind and will constitute a changing content of consciousness, just as is the case in abstraction, the state commonly used to obtain so-called free associations.

Amnesia.—Absence of memory for the hypnotic experience may or may not follow on light hypnosis. The same is true of abstraction, mutatis mutanda.

After obtaining free associations in the ordinary state of abstraction, I have occasionally noted extensive amnesia for certain memories on "waking," and I have no doubt that others have also. Whether or not there is amnesia following such light dissociated states depends, in my judgment, on various factors and particularly on the degree and extent of the dissociation of the personality and possibly on the intensity of the previous repression of the memories from conflict.

Suggestibility.—Much has been made of the increased suggestibility in hypnosis, but whether there is a greater degree in the light stage under consideration than in some types of abstraction remains to be proved. Crystal visions and automatic writing are common suggested phenomena in normal abstraction. If suggested contractures are taken as a criterion, as is commonly done, they can often be produced in the normal alert state. Coué, in his public demonstrations, has notoriously induced them in apparently alert subjects. Various observers have reported striking examples of contractures produced in alert subjects. I induced strong contractures in an alert subject by using a tuning fork, which, to

impress her, was represented to be a powerful magnet. On the other hand, it must not be overlooked that probably only in a minority of cases of light hypnosis can contractures be induced. They belong more characteristically to somewhat deeper stages of depersonalization. Likewise, suggested analgesia is a common phenomenon in alert subjects in expert hands.

Comment.—When the phenomena of hypnosis are considered from this point of view as phenomena of integration, dissociation and inhibition, it would seem plain that the state is one of depersonalization and repersonalization. Personality, according to the theory that offers the more intelligible and adequate explanation, as I have frequently argued on the basis of studies of abnormal as well as of normal conditions, is an organized integration, on the one hand, of the sensory and motor functions and, on the other, of the innate instinctive dispositions—the so-called emotional "instincts"—plus the dispositions and systems of dispositions acquired through the experiences of life. All these, speaking roughly, include the "traits" of the individual. These traits consist of the emotional urges and tendencies, the habit reactions, the sentiments, ideals, beliefs, etc. Obviously, so far as and so long as any of these components of personality are dissociated from the whole integration, there is a depersonalization. And so far as and so long as there is a new and different integration of the remaining components, there is a repersonalization.

A tabulation of the negative phenomena observed in hypnosis, then, shows that there is a dissociation or inhibition of many of the component traits and reactions of the normal alert personality resulting in what one is entitled to call depersonalization.

Between the light phases of hypnosis and the deep phases (somnambulism) there is an infinite variety of phases differing from one another only in degree and complexity of dissociation and integration.

The next point I want to make is that light hypnosis is substantially nothing more or less than abstraction, or at least a type of abstraction. There are several types of abstraction, all conforming to the same principles of integration, dissociation and inhibition and differing only in the structure of the final integrate, according to the source and specificity of the stimulus (suggestion, intrinsic interest of the subject, endopsychic stimuli, instinctive impulses, etc.). No two types and no two states are precisely the same in respect to the elements of personality that are integrated and dissociated.

If this is true and light hypnosis is a type effected artificially by suggestion, one must not forget that it is an artefact, and that therefore the type and its phenomena are artificially determined and tend to conform to the suggestions given.

It would follow from this interpretation that any physician who uses passive abstraction for the purpose of obtaining free associations in psychoanalysis (as every one does) or suggestive therapeutics uses hypnosis.

Again it follows that, as every one can be induced to go into a state of passive abstraction, every one can be repersonalized (hypnotized) to this extent.

Hypnosis, then, as I said at the beginning, does not stand apart as a bizarre condition of mind and body; it is one type of a large category of conditions, the essence of all of them being a normal, artificial, or abnormal alteration of the structure of personality. All types are the resultants of the same processes and are characterized clinically by depersonalization and repersonalization of the whole structure of personality. The differences of type are brought about by differences in the motivating impulses that effect dissociation and integration. The only and insufficient reason for differentiating hypnosis from the general category is that it is a suggested artefact, though this is not always the case.

In view of these principles and the facts of observation, I have suggested the term "repersonalization," or "suggestive repersonalization," in place of hypnotism. The former correctly characterizes the facts; the latter does not, but it expresses an outgrown theory that identifies hypnosis solely with sleep and neglects other allied forms of altered personality.

This point of view—depersonalization and repersonalization—is important and fruitful in that it allows one, on the one hand, to relate hypnosis to fundamental principles governing the functioning of the mind and, on the other, to class it in a large category of normal states such as sleep, abstraction, revery, mystic ecstasy, moods, absentmindedness and emotional crises, and abnormal states, such as trance, hysterical crises, fugues, somnambulisms and double personalities.

II. THE MOTIVATING FORCES

Although it is not strictly within the scope of this paper, I feel that some consideration of the forces that induce suggestive repersonalization is called for, as the question is sure to be raised. Several theories have been offered but in my judgment none can be said to have been substantiated.

All students agree, I believe, that the phenomena are induced by suggestion in some way. It is therefore around the nature of suggestion, the nature of the force or forces which it evokes, and the mechanisms which are brought into play by those forces that the discussion revolves as the real problems.

According to the original freudian hypothesis, the essential characteristic of hypnosis and suggestion is a dissociation of consciousness (an assumption that I have tried to show is inadequate), but this dissociation is not primarily, as commonly believed, "an artificial state brought about by the hypnotic procedure. The dissociation is already present for the operator to make use of." [2] It exists in the form of "infantile incestuous thoughts" in the unconscious (the mother or father complex) which have been repressed from infancy; that is, ready to be fixed on the operator. The essence of hypnosis resides in the unconscious fixation of sex hunger (the libido) on the person of "the hypnotizer." And the "capacity to be hypnotized and influenced by suggestion depends on the possibility of transference;" i. e., on the "unconscious sexual attitude" of the hypnotized to the hypnotizer—the deepest root being in the repressed parental complex (Freud, Jones, Ferenczi and Sadger). When this is translated into ordinary everyday language, it means that the subject unconsciously becomes sexually in love with the hypnotizer. By what mechanism the phenomena of hypnosis are induced because of this fixation is not clear. After as critical a study as I have ever given to anything, the reasoning by which hypnotic phenomena are induced on this theory of "transference" seems to me a mere hodge-podge of logic. It is useless to discuss it here; yet I think that this sexual theory may be modified and restated in a form to give it a certain plausibility, although it would no longer be freudian.

In the language of the psychologist, there is a response to the stimulus (suggestion) of the hypnotizer on the part of the subject, in the form of an unconscious reaction of the "libido" contained in the repressed infantile thoughts. The urge of this libido, then, becomes unconsciously integrated with the suggestion given and supplies the impulsive force or drive that carries the suggestion to fruition; that is to say, produces the phenomena of dissociation, inhibition and integration and finds satisfaction.

Even when so formulated, the theory is, to my mind, unsound for many reasons. Certainly, if the thesis for which I have argued here is true, this modified freudian hypothesis is incredible, for surely, if, as I have insisted, hypnosis is only a type of repersonalization not differing in principle from abstraction in its several forms, sleep, trance, spontaneous somnambulism, etc., it is almost unthinkable that sexual love induces all of these common and (some of them) everyday phases of personality.

The most commonly advanced theory (called by McDougall the "ideomotor" theory) holds that the suggestion of the operator awakens

2. Jones, Ernest: Papers on Psychoanalysis, William Wood & Co., New York, 1919, p. 321.

as a reaction an "idea" in the subject. This "idea" (which is a representation of the state expected by the subject), reinforced by any emotion organized within it as a sentiment, has in itself an adequate driving force to bring about dissociation (and integration?) and therefore the phenomena corresponding to the representation in the consciousness of the subject.

McDougall rejects this theory on the ground that so-called "ideas" have no motive force of their own, but derive their impulses solely from becoming linked up with one or more emotional instincts. He proposes, accordingly, what is really a necessary corollary to and application of his theory of the nature of instincts and the purposive part they play in mental behavior. His explanation of suggestion and rapport therefore demands careful examination.

To appreciate the full meaning of his theory, it must be borne in mind that, according to him, "ideas," so-called, are of themselves lifeless, inert things, incapable of carrying themselves to fruition. To become effective, they must become linked up with one or more of the emotional instincts (fear, anger, curiosity, etc.). The drive, urge, impulse, or craving of these instincts provides the driving force that renders the idea capable of being carried to fulfilment in accordance with the aim of the instinct. Among his instincts is that of submission or self-abasement. It is this instinct, in his view, that is always awakened by the hypnotizer and thereby, becoming linked with the suggested idea, gives the suggestion the force which induces the dissociation of the personality on the one hand, and creates the submissive "rapport" of the subject with the hypnotizer on the other.

The reason for the awakening of this particular instinct is to be found in the attitude of mind of the subject toward the hypnotizer. He looks up to the latter as a being of superior power whom he endows with prestige, authority and knowledge and therefore to whom he feels inferior. In fact, this instinct is the basis of the feeling of inferiority—the so-called "inferiority complex." I am ready to accept the facts that there is such an instinct with a "drive," even if not of the purposive nature attributed to it by McDougall, that it is awakened in many cases of hypnotic suggestion, and that it then provides the main driving force to the suggestion, or at least cooperates in doing this.

However, my objection to the theory is that it is overworked. A priori, there surely is no reason why other instincts may not be equally effective. I am unable to accept the statement that the submissive instinct is universally applicable to all instances of hypnotic suggestion, or to all, or many, suggestions in everyday life (which must also be explained); or that in many cases of hypnotic suggestion the awakened and efficient impulse is not that of another instinct or interest, such as self-assertion, curiosity (scientific curiosity), or some such inter-

est as apparently is the effective force in normal abstraction, or that it may not be faith or expectancy. It also leaves out of account hypnosis induced by autosuggestion, contagion, and even by a secondary coconscious process or personality.

Many persons can hypnotize themselves or learn to do so. Mediums hypnotize themselves, producing the so-called trance state, which is identical with hypnosis, as shown by the fact that it is often produced by "hypnotic" suggestion.

Furthermore, the fact is significant that hypnosis can be induced in the primary personality by a coconscious secondary personality. It may be recalled that Sally, a coconscious personality, repeatedly hypnotized the principal personality, Miss Beauchamp, by coconscious "willing."[3] Also in the case of B. C. A., a coconscious intelligence repeatedly induced a trance state in the principal personality.[4] Is one to infer in such observations that the hypnosis was induced by the force of the instinct of submission, notwithstanding the fact that she was unaware of the coconscious personality and could not know that she was being hypnotized by herself in this bizarre fashion?

Human motives are too complex to be reduced in all cases to one simple instinct, whether it is sexual, submission, fear, or any other.

Nor will any instinct, though it may be the driving force of the suggestion, explain all the phenomena that characterize the hypnotic state, such as many of those I have marshaled in the foregoing as phenomena of dissociation and integration (anesthesia, paralysis, contractures, somnambulism, hallucination, etc.).

It seems to me, also, that ideas have an intrinsic driving force in themselves, however much this force is reinforced and strengthened by the more powerful forces of the emotions; consequently, I cannot wholly reject the ideomotor theory.

That the force of the suggestion inducing hypnosis may be derived not from the instinctive emotion of submission but from that of self assertion is shown by the case, as I interpret it, of Mrs. J., which I cited as an example of epochal amnesia.

When I first attempted to hypnotize her, it was without the slightest success. Whereupon she turned to me and said, with some strong feeling of self-assertion and pride, "No one can hypnotize me. My will is stronger than any one's will. You can't do it." Thereupon I said, "You are right. I can't. But the reason is you are afraid. You have not the courage to be hypnotized; you are a coward; you haven't the 'sand.'" At once, as if to show me not only that she was not a coward

3. Prince, Morton: Dissociation of a Personality, Longmans, Green & Co., New York, 1906, pp. 319.

4. My Life as a Dissociated Personality, by B. C. A., J. Abnorm. Psychol., October, 1908, and December, 1909.

but also that she could do anything she willed to do, she promptly went into the deep somnambulistic state I have described. In this state, it will be remembered, she reverted to a previous period in her life with epochal amnesia for the nine succeeding years. She also exhibited a delusional phenomenon which is not uncommon in such conditions of reversion. I have observed it often. At the period to which she reverted, she was relatively slim and slight, with a corresponding waist measurement. When I showed her the tape measure recording the increased size of her waist, she insisted that the figures were several inches less, reading them in accordance with her illusion of living at the time when that was her girth.

Even if her attitude at the outset was bravado and a reaction to the submissive instinct, the motivating force must have been the instinct of self-assertion.

However, whatever the driving force is that gives effect to suggestion —whether it is derived from the sexual impulse or that of the instinct of submission or self-assertion, or any other—no theory so far advanced will give an adequate explanation of all the phenomena of hypnosis and especially of the integration, though it may explain the simpler phenomena of dissociation, particularly of the lighter stages. Nor will it throw light on the more important problems of the mechanisms by which many phenomena are effected, even such simple ones as automatic contractures. Why, for instance, was the hypnotic state in the case just cited characterized by retrograde amnesia and the delusional phenomena? Whether or not the instinct of submission or self-assertion was the motivating force, it will not explain these and other specific phenomena.

Likewise, many of the more complex phenomena of integration are left totally unexplained and require some other principle. McDougall himself admits that a number of phenomena remain obscure.

Let us take, for example, somnambulism with the integration of large systems resulting, without apparent rhyme or reason, in a secondary personality with the loss or dissociation of particular instincts, and other functions. To explain such phenomena one must, it appears to me, introduce the principle of conflict, as I have done when interpreting the psychogenesis of multiple personality. The suggestion that the subject be hypnotized (i. e., "sleep") for some reason, obscure at the time, strikes some sentiment or other psychologic component in a large integrated system (in B. C. A., as elsewhere, I have endeavored to show [5] a rebellion against the conditions of life) and thereupon the whole system, in conflict with the rest of the personality, springs into

5. The Unconscious; The Macmillan Co., New York, 1919, Lectures 18 to 20; also J. Abnormal Psychol., Oct., 1919.

life and becomes the secondary personality. Through conflict, the rest is repressed. But why should certain fundamental and native instincts like fear, anger or sex become dissociated? This is a riddle.

And why in the same subject in one hypnotic phase should there be a dissociation of one set of traits and certain functions with integration of certain other specific traits and functions, while in a second and third phase the dissociations and integrations are entirely different? Why, for instance, in one phase of hypnosis is there general anesthesia; in another, anesthesia of a special sense, and in a third no anesthesia at all? This applies to other functions and mental traits (sentiments, etc.). The suggestion in each case was identical, being simply "sleep" without any specific implication and expectation of the resulting phenomenon, which was a surprise. Here one is dealing with mechanisms and forces that are totally obscure. One must look for endopsychic forces as explanations, and among them internal conflicts between mental systems.

These and more phenomena are unexplainable by any theory that neglects to take into consideration all the phenomena of hypnotism, particularly the integrations and their motivations, as well as the same phenomena occurring in allied states like abstraction, sleep, trance, double personality, etc.

DISCUSSION

Dr. Sidney I. Schwab, St. Louis: What are these absolute tests for depersonalization? How does one know when a person is depersonalized? Are there any phenomena when the various personalities come in conflict with each other? What are the objective evidences of these things, not something that is in one's own mind about them, but what do the patients show from an observation point of view that would lead one to deduce these facts?

Dr. Smith Ely Jelliffe, New York: I feel very much in sympathy with Dr. Prince with reference to the difficulties of definition. One need not necessarily review the history of Greek philosophy to realize that Heraclitus said that everything was in a state of flux, nor that Zeno introduced a knotty series of philosophic problems which Dr. Prince has touched on, namely, the difficulties of clearly formulating various levels whereby descriptive terms may make one able to handle them. From the epistemologic point of view, I am a great deal in sympathy with the general thesis that Dr. Prince has worked out.

I should have liked it if he had touched more on certain aspects of the dynamic situation, because just as it is difficult to define personality, just so it is difficult to define what is meant by conscious, foreconscious and unconscious. The whole problem of hypnosis and its related series of phenomena is evidently closely associated with the problems that arise when one attempts to formulate what one would like to mean by foreconscious and unconscious phenomena.

I think that if, for the time, I should choose to be ultradidactic, such a formulation as follows might be hazarded: that everybody regards everybody else from two aspects. The deeper in the foreconscious one goes, the more apparent become those two aspects. These are the father aspect and the mother aspect. There is a dynamic relationship between everybody and everybody else viewed as father and as mother images. The deeper one enters into the unconscious, the more the identifications with father and with mother take place. The series of phenomena

that Dr. Prince has briefly indicated as split-off bits of inhibition, as bits of a dissociation, from such a point of view, I think, can be formulated as bits of special experience that one has lived through at various stages in his childhood. Then later, one's attitudes toward his father and toward his mother become extended to other relationships.

One knows that when it comes to the larger question of mass psychology such as, for instance, this meeting (which is an illustration of mass psychology), the same dynamic relationships exist. With what attitudes do we regard one another? What various optical reflex attitudes are we going to place ourselves in with reference to the auditory stimuli, i. e., the various forms of symbolic meanings? What is going to be your father-mother attitude to such symbols at the conscious level, at the foreconscious level and at the unconscious level?

And so it would seem to me that the larger problems that Dr. Prince has touched on have an enormous significance in life in every way, shape, manner and form, and that the hypnotic portal is one of the ways by which such problems may be entered.

There is only one point of dissent that occurs to me now with reference to what Dr. Prince speaks of. That is the possibility of reintegration through the hypnotic process. In the discussion of education, emphasis has been laid throughout on the building up of the conscious attributes that are necessary to such educational processes. From my point of view at least, it seems that the hypnotic system, or the hypnotic slicer of the personality, does not provide therapeutically that series of conscious continuous processes that permits an integration. Therefore, it has seemed to me that it does not fill the bill psychotherapeutically. For me it never has, and that is one of the reasons why I have never done much with hypnosis and why I am so little qualified to talk about the matter.

Dr. Hugh T. Patrick, Chicago: I should like to express my feeling about this interesting presentation. That hypnotism is generally misunderstood and misapprehended is certainly true, but I do not see any reason why one should adopt repersonalization and depersonalization for what has heretofore been known as hypnotic procedure. That hypnotism consists in part of depersonalization and repersonalization is sufficiently acceptable to me. But what is miscalled hypnotism or poorly called hypnotism is, after all, only a small fragment of depersonalization and repersonalization as they are seen day by day.

All psychotherapeutic procedures are really attempts at depersonalization and repersonalization. Whether it is by suggestion, by what might be called reeducation, by discipline, by encouragement, by persuasion, the attempt always is to produce inhibition, integration and reintegration; in other words, to tear down some parts of the personality and build up others. If that is not a process of (attempted) depersonalization and repersonalization, what is it? Hence, why reserve for this thing that has always been called hypnotism the term "repersonalization?" It would be restricting to a narrow field a term in the nature of things of general, almost universal application. Consequently, I think that Dr. Prince's proposal is not practical or feasible and is quite illogical.

Dr. C. P. Oberndorf, New York: As Dr. Prince has so well pointed out, the gradations between hypnosis and normal mental phenomena, such as abstraction, are obvious to any one dealing with psychopathologic states. As is well known, psychoanalysis sprang directly from hypnosis. Freud, following the French school, first used hypnosis to gain access to the unconscious. After studying with Charcot, and during his subsequent visit to Bernheim's clinic in Nancy, he was much impressed by the fact that many patients refused to be hypnotized by Bernheim, whereas others were readily accessible. This led him into an inves-

tion, as Dr. Jelliffe said. The reason that a certain patient is open to extraordinary tigation of the reasons why certain persons were open to hypnosis and others were not. This is a cardinal point in the consideration of hypnotic phenomena and one that Dr. Prince has not broached. Freud soon came on the mechanism of identification and concluded that suggestibility is entirely a question of identificasuggestibility at the hands of one person and not another, or one method of hypnotizing succeeds when another method fails, is because of an unconscious identification by the patient of the particular hypnotizer with his father or his mother and their manner.

Dr. S. Philip Goodhart, New York: In a less controversial spirit and tone, due perhaps to a less militant personality, I wish to express my approval of Dr. Patrick's attitude toward Dr. Prince's presentation. I feel that the application of the terms "depersonalization" and "repersonalization" is altogether too general. I would limit the application of these terms to the cases he has mentioned, namely, to those that show a real splitting of personality with a manifest change in character and behavior, in which there was especially a change of mental attitude toward life. I refer particularly to instances of dual or multiple personality in which there really is a depersonalization—a removal to a considerable degree of the individual's psychologic attitude with its replacement by perhaps a widely different one. In these cases, as a rule, amnesia divides the two personalities; the old may be repressed and a new one come to the surface. Thus a depersonalization and repersonalization may be spoken of. The minor and temporary variations such as may be induced by hypnosis are such fragmentary elements of a personality that to dignify them with the appellations suggested by Dr. Prince would tend to mislead us in our understanding of their psychology.

Dr. Prince: In regard to the questions asked by Dr. Schwab: How do we know that a person is hypnotized or depersonalized, and what are the objective evidences of that fact? In the lighter stages that is difficult to determine, if one is going to hold that hypnotism is a specific, definite state. I tried to bring out that it is not a specific, definite state, but is only a type (in its light stages, I mean) of abstraction. I might ask, then, how does one know a person is in abstraction? How does one know he is absentminded? How does one know he is in a state of revery? A person knows for himself when he is in that condition. But one has, more or less, to take his word for it. When the hypnotizing process is carried a step further and a deeper state is obtained, it presents an interesting problem. What has happened? I have often said to such a subject, "Now, tell me when you feel you are hypnotized." The subject may answer "Not yet, not yet" —and then in a few moments, "gone," he will say. "Now tell me how your present state seems to you to differ from your normal state awake." I have never been able to obtain such a description that would enable me to identify precisely the subjective characteristics of the state. The best conclusion I could come to was that the person has felt a dissociation, a physiologic dissociation of the body; it seemed to be a definite psychologic condition, recognizable by the subject.

However, my point is that there is not, in the light stage, any distinguishing difference between it and normal abstraction. It is only when one goes deeper and deeper that he finds this difference from normal waking states. But it is one of degree of dissociation and integration. One finds every grade of dissociation and integration from abstraction up to these extreme stages of repersonalization.

It is known, of course, that there is no difference between a somnambulistic stage brought about artificially by hypnosis and one that is spontaneous or brought about by autosuggestion or endopsychic processes. That is why I took these more obstrusive extreme types first, in order that one should become familiar with the

phenomena of dissociation and integration. It is necessary to know the phenomena before one can understand these processes and form opinions about hypnosis. One must be familiar with all the phenomena, of which there is a great number and variety.

The point I wanted to make was that the general conception of hypnosis or the hypnotizing process is dissociation. Every one lays stress on dissociation but overlooks the integrating process. Nothing shows the integrating process more beautifully, I think, than the technic that I have used for reintegrating a double personality. I have used it in several cases with success.

For example, there are two secondary personalities, *A* and *B*, each with distinct traits, distinct memories, distinct sentiments, distinct instincts, emotions, etc. —entirely different personalities. Now I hypnotize *A* and get a hypnotic state, which is essentially the same in all its traits, as the waking state, *A*, but a passive dissociated state characteristic of a moderately deep state of hypnotism. Then I hypnotize *B* in the same way, using in both cases simply the formula "sleep," and obtain a dissociated state identical in traits with *B*. As with *A* it is *B* hypnotized. Now in each case I go on further and say, "Sleep deeper, deeper," nothing more than that. What happens? Both hypnotic *A* and *B* change to one and the same hypnotic state *C*, which has all the traits of *A* and *B* combined, integrated. I only said, as before, "sleep" but in place of dissociation I obtained integration. I only said to both *A* and *B*, "Sleep deeper," and yet the process was one of integration, not dissociation, resulting in one and the same state which is the whole personality, hypnotized. Then all I had to do was to awaken this one (*C*), and I obtained the total integrated personality, a combination of the two; I had integrated them by the hypnotizing formula "sleep."

The principle of integration is likewise illustrated by the two cases of Jules Janet. In one case there was hysterical vomiting so severe as to threaten the life of his patient; she also had chorea. Now, Janet merely hynotized that person, told her to sleep. She went into a different state and became normal and free from vomiting and other symptoms; it was her normal personality. But it was mistaken by Jules Janet for an hypnotic state. It was not. It was the normal personality. By the process of hypnotizing, he integrated that personality.

Dr. Jelliffe has raised probably the most important, the most interesting, at least, question, which I did not touch for lack of time, although I have discussed it in my full paper. That is, what is the nature of suggestion? What is the force, or what are the forces which motivate the suggestion to bring about these alterations of personality, these dissociations on the one hand and integrations on the other? I wish I could have gone into it.

There are various theories. There is the freudian theory, the ideomotor theory, the theory of McDougall, to which I referred, and there are others. In my judgment, none of those theories has been substantiated or is wholly adequate. What one wants to know first is not so much what the forces are but what are the mechanisms by which these extraordinary phenomena—contractures, amnesias, etc.—are brought about. In my judgment this is not known. It is a field that must be further investigated. It is much better to say, "we do not know," than to propose inadequate theories. I think little is known about it. Undoubtedly in some instances conflicts are aroused in the mind which awaken whole systems of experiences, memories and impulses, etc., and which come in conflict with other systems and produce these extreme dissociations; but it is a field, I think, in which a great deal more study must be made before one is able to dogmatize and have theories that cover all the phenomena.

Dr. Patrick has raised a point with which I sympathize. Artificial repersonalization by suggestion does not differ in principle from repersonalization and depersonalization in many and varied phases of everyday life. I tried to make that clear. In other words, as I said in the beginning, the same processes—dissociation, inhibition, etc.—are at work in various forms in normal everyday life. I also tried to make clear that, in my opinion, the hypnotic state does not differ in principle from various abnormal states. These two points are fundamental to my main thesis, the nature of hypnosis. I am surprised that this was not understood. I think, nevertheless, that a different and better term than hypnotism should be used for suggested repersonalization for reasons I have given. Perhaps the term "suggestive repersonalization" would meet the objection raised by Dr. Patrick and I shall be glad to add the qualification "suggestive" to define this particular type of repersonalization.

POSTCONCUSSION NEUROSIS—TRAUMATIC ENCEPHALITIS

A CONCEPTION OF POSTCONCUSSION PHENOMENA *

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Psychogenic or organic? Which is the determining factor, at least in the beginning, of what is commonly called the postconcussion neurosis? When these patients come to the neurologist the symptoms usually are chiefly, or exclusively, subjective. It is difficult to say whether the symptoms complained of are purely of emotional, and therefore psychogenic, origin. The trend of the neurologist's thought in this connection is apt to depend on whether he has learned to stress the psychologic factors or whether, on the other hand, he seeks the structural, organic causes. Our work shows that the structural factors in postconcussion neurosis have not received adequate attention.

We were impelled to make this study by two facts. Early in our observation of cases of concussion, we became impressed with the difference in the nature of the reactions from cases of posttraumatic psychoneuroses in which it seemed fair to assume that the injury of the head, if one occurred, did not result in actual disturbance of tissues of the brain, as evidenced chiefly by unconsciousness. These differences also included certain objective symptoms present in the concussion group which were absent in the psychoneurotic group. Equally decisive differences, it seemed to us, existed in the nature of the purely subjective complaints. Many of the symptoms in the postconcussion cases were so like some of those complained of by sufferers from the residuals of epidemic encephalitis that the resemblance at times was startling.

MATERIAL

Our material consists of 100 cases of concussion of the brain, with or without fracture of the skull. In sixty-eight there was a wound in the scalp existing either alone or associated with fracture; in thirteen cases it was associated with fracture of the skull at the base, and in thirty-six cases with fracture of the vault, leaving ninteen cases in which

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the only evidence of injury was the wound in the scalp and thirty-two cases in which not even a wound in the scalp was present. In fifty-three of the 100 cases there were injuries in parts of the body other than the skull. Table 1 shows in detail the statistical data referring to the material. Ninety-six of the patients were men, two were children, and two were women.

The patients were injured mostly by falling while at work; some had been struck by various objects; a few, knocked down by automobiles, and several struck by blunt objects during an assault. All were claimants for damages for the injuries received, either under the compensation laws of this or neighboring states, or under the laws covering liability. In all cases the history was confirmed by reference to hospital records or by statements of reliable witnesses, and the essential symptom of unconsciousness at the moment of injury was definitely verified

TABLE 1.—*Statistical Data Referring to Material*

External Evidences of Injury:	
A. Scalp	68
B. Fracture of skull base	13
C. Fracture of vault	36
D. Other injuries	53
Bleeding from Orifices:	
A. Ears	23
B. Mouth	21
C. Nose	20
D. Ecchymosis about eyes	4
Sensory Disturbances:	
A. Functional type	21
B. Organic type slight (cortical type as described by Head)	2
Decompression Operation:	
A. Not for depressed fracture	4
B. For depressed fracture	6
Functional paralysis	1
Pyramidal tract signs	6

before we included the case in this series. This was the only type of selection used in gathering this group. The presence or absence of fracture of the skull was determined by competent roentgen-ray investigation. Many of the patients were seen several times and some were kept under observation for varying periods. The 100 cases of epidemic encephalitis used for comparison of the subjective symptoms were studied in our private practice in recent years.

DEFINITION

Since Trotter's[1] definition of the term concussion seems to be generally accepted, we quote it verbatim:

> I may say at once that I use the term concussion, as I think it should only be used in the strict classical sense, to indicate an essentially transient state due to head injury which is of instantaneous onset, manifests widespread

1. Trotter, Wilfred: Certain Minor Injuries of the Brain, Lancet **1**:935 (May 10) 1924.

symptoms of a purely paralytic kind, does not as such comprise any evidence of structural cerebral injury, and is always followed by amnesia for the actual moment of the accident.

There are two exceptions to be made to this definition in view of what we will have to say later: (1) It is not possible to say in a given case of concussion, "that the state is essentially transient," unless one recognizes that these words simply apply to the unconsciousness and the immediate paralytic phenomena which accompany or follow the unconsciousness. (2) It is not tenable, in the light of what will follow, to advance the proposition that concussion of the brain "does not as such comprise any evidence of structural cerebral injury." With these two exceptions, we are prepared to subscribe to Trotter's definition of concussion.

PATHOLOGY

Many textbooks on surgery, nervous diseases and pathology are at one in saying that the pathology of concussion of the brain is not definitely known or does not rest on solid ground. Typical of this point of view are the statements to be found in the textbook on legal medicine by Peterson, Haines and Webster [2] that "nothing definite is found in concussion and the general cerebral congestion is also found when death is due to alcohol." The reason for this is the fact that few persons die from simple concussion of the brain and the number that reach necropsy are so rare that the literature is practically barren.

Most of our knowledge of the pathology of concussion is deduced from a study of more serious injuries of the brain. There have been a great many studies of craniocerebral injuries, both on the living patient and on necropsy material. The most important of these is the result of the war experiences of Trotter,[3] Head,[4] Shields,[5] Riddoch,[6] Cushing,[7] Jefferson,[8] Duperié,[9] Hine [10] and Mott.[11] Similar studies in

2. Peterson, Haines and Webster: Legal Medicine and Toxicology **1**:309, 1923.

3. Trotter, Wilfred: Shell Wound of the Head, Brain **42**:353, 1919.

4. Head, Henry: Shell Wound of Head, Brain **42**:349, 1919; Sensation and the Cerebral Cortex, ibid. **41**:57, 1918.

5. Shields, Oswald: Gunshot Wound of Head, Brain **42**:355, 1919.

6. Riddoch, George: Case of Meningitis Circumscripta Serosa Following a Bullet Wound of the Neck, Brain **42**:360, 1919.

7. Cushing, Harvey: A Study of a Series of Wounds Involving the Brain and Its Enveloping Structures, Brit. J. Surg. **5**:565, 1918.

8. Jefferson, Geoffrey: Gunshot Wounds of the Scalp, Brain **42**:93, 1919.

9. Duperié: Récherches sur les symptomes homolateraux dans les perforations du crane et de l'encéphale par les projectiles de guerre, Rev. neurol. **29**:616, 1916.

10. Hine, M. L.: Recovery of Fields of Vision in Concussional Injuries of the Occipital Cortex, Brit. J. Ophthalmol. **2**:12, 1918.

11. Mott, F. W.: War Neuroses and Shell Shock, pp. 4, 5 and 38 to 66, 1919.

this type of injury occurring in civil life have been made by Adolf Meyer,[12] Cassasa [13] and others. The pathology of concussion of the brain and of injuries of the brain in general is also discussed by Adami,[14] Adami and Nicholls,[15] and Charles Foix.[16] The late anatomic results of concussion and of injury of the brain in general have been studied by Adolf Meyer,[12] Foix,[16] and Tanzi and Lugaro.[17]

MECHANISM

Adami [14] points out that the cerebral blood vessels, having a slight muscular coat, rupture easily because the substance of the brain offers little support. This observation is of importance in considering the explanations of the mechanism of the production of concussion. Those of Mott and Cassasa complement each other. Mott [18] says, "The stem of the brain, surrounded by the cerebrospinal fluid, is prevented from oscillating by the nerves which issue from it to pass through the holes in the skull; likewise the spinal cord, by the anterior and posterior roots and the ligamentum dentatum, is prevented from oscillating. A sudden shock of great intensity would be transmitted through this incompressible fluid, and, seeing that it not merely surrounds the central nervous system but fills up the ventricles and central canal and all the interstices of the tissues, serving as it does the function of lymph, it follows that a shock communicated to the fluid of sufficient intensity would make itself felt on all the neurons." Cassasa's explanation [19] of the mechanism of concussion depends on the identification of "a net work of fine fibrils connecting the external wall of the blood vessel with the surrounding brain tissue across the perivascular lymph space." Figure 1 illustrates this point.

Sudden overfilling of the perivascular lymph space with cerebrospinal fluid conceivably could produce laceration of a vessel by tearing of its wall in the neighborhood of such a fibrillar attachment. Otherwise, without such an attachment, the laceration of a vessel surrounded by fluid could not be produced by any pressure exerted through that fluid which would only tend to

12. Meyer, Adolf: The Anatomical Facts and Clinical Varieties of Traumatic Insanity, Am. J. Insan. **6**:374, 377, 382 and 388 (Jan.) 1904.

13. Cassasa, C. B.: Multiple Traumatic Cerebral Hemorrhages, Reprinted from Proceedings of the New York Pathological Society, N. S. **24**:101 (Jan.-May) 1924.

14. Adami, J. G.: Principles of Pathology **1**:282, 1910.

15. Adami and Nicholls: Principles of Pathology **2**:595, 1911.

16. Foix, Charles: Traumatic Lesions of the Skull and Brain. Diseases of the Nervous System, in Nelson's Loose-Leaf Living Medicine **6**:145-J, 1920.

17. Tanzi and Lugaro: Malattie Mentali **2**:325, 1914.

18. Footnote 11, p. 4.

19. Footnote 13, p. 102.

compress the vessel but not lacerate it. Such an increase of cerebrospinal fluid in the perivascular lymph space could be caused by the cerebrospinal fluid from the surface of the brain being driven into it by the pressure exerted by the change of shape of the skull—the result of a blow or fall. This change of shape under an area of violence is in the direction of flattening and diminution of space for the cerebrospinal fluid in that area. This fluid must find its way out of that area through the various sulci of the brain and in connection

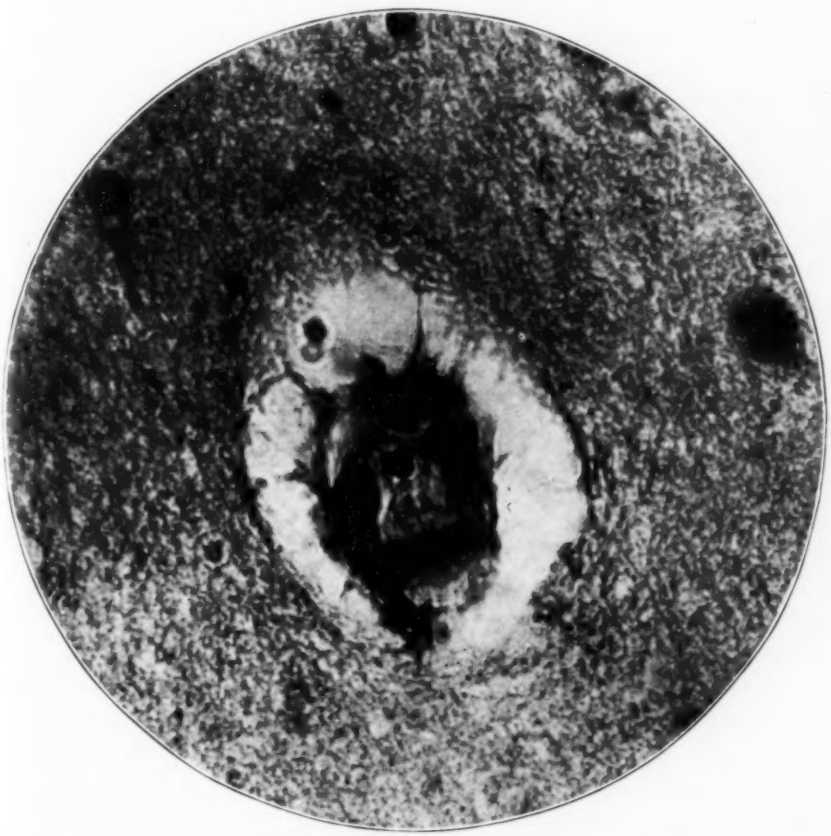

Fig. 1.—Network of fine fibrils connecting the external wall of the blood vessel with the surrounding brain tissue across the perivascular lymph space.

therewith such fluid as cannot find its way through these channels must find a way into the perivascular lymph spaces in the reverse direction of the normal flow of the cerebrospinal fluid in these channels.

Cassasa then refers to the experiments of Weed which demonstrate that the perivascular lymph sheath (Virchow-Robin space) is directly connected with the finer spaces about the cerebral ganglion cells. He now advances the theory that sudden physical distention of these peri-

ganglionic spaces by cerebrospinal fluid, in cases of concussion, causes a direct physical change in the ganglion cell which may explain all the phenomena of concussion of the brain. This simple change may occur without minute hemorrhages and explains the completely negative observations of some authors who have performed necropsy in cases of concussion.[20]

PATHOLOGY OF INJURIES OF THE BRAIN

Trotter [3] and Head [4] have described the living pathologic changes in certain cases of traumatic epilepsy, and in cases of persistent headache in which operations were performed a long time after shell wounds of the skull. Trotter noted that the arachnoid is apt to be opaque and slightly distended by fluid collected under it. The brain is stained orange as a result of extravasation of blood. This appearance is said to be typical of old unresolved contusion. These authors lay a great deal of stress on the conception that the brain undergoes considerable swelling as a result of injury at the site of contusion, and that this edema accounts for the increased intracranial pressure and for many of the persisting symptoms. This conception was originally worked out by Cannon.[21]

The pathology of craniocerebral injuries is summarized by Jefferson.[8] This author's observations are of interest because he dealt exclusively with gun shot wounds of the scalp in which there was not any demonstrable injury to the skull. Most of his cases, therefore, were of simple concussion. Fifty-four cases came under his care in a general hospital in England during the World War. At operation, there were definite areas of contusion of the motor cortex in eleven, of the visual in four, and of the motor sensory cortex in five. Jefferson also confirmed the presence of contralateral contusion of the brain by contrecoup in four cases, an observation previously referred to in the series published by Duperié.[9] The gross appearances were merely slight meningocortical hemorrhage. By lumbar puncture, clear and untinged cerebrospinal fluid was obtained.

In the more severe cases of injury of the brain described by Cushing,[7] Duperié [9] and others, there is actual disorganization of the substance of the brain with severe intracranial hemorrhage.

LATE PATHOLOGY OF INJURY OF THE BRAIN

Meyer [12] and Tanzi and Lugaro [17] were pioneers in describing the residual distinctive changes of cerebral trauma. According to Meyer, they are small foci of softening or defects of cortex in the tips of the

20. Personal communication from Dr. Cassasa.

21. Cannon, W. B.: Cerebral Pressure Following Trauma, Am. J. Physiol. **6**:91, (Oct. 1), 1901.

frontal or temporal lobes with the absence of glia margin beneath the pia but also an increase (broadening) of the sub-pial glia in the same brain. The diffuse effects are extensive. One of the cases showed extensive lesions involving many parts of the brain but particularly degeneration of the "median striae Lancisii and destruction of both olfactory bulbs. The optic chiasm showed unmistakable semidecussation, and the indirect scattered lesions, through the concussion, were exceedingly extensive and involved mainly the splenium of the corpus callosum and the long paths—the fillet and the superior cerebellar arm. There was also slight implication of both fifth nerve roots and of the optic nerves." While Meyer felt that the diffuse effects were due to concussion, it is well to note that case 5 of Meyer's series, which particularly brought out these observations, was one in which there was a gross craniocerebral injury in the left frontal region, with prolapse of brain fungus into the skin, which later developed infection, and that, although there was not any meningitis, pus was found in both lateral ventricles. In other words, even in Meyer's series the same difficulty is found that was encountered in studying the cases resulting from war trauma in the series reported by Cushing,[7] Head [4] and others, namely, the injuries to the brain were much more severe than we have reason to believe occur in simple cases of concussion. Tanzi and Lugaro appear to attribute a purely psychogenic origin to the cases of what they call traumatic neurosis, but in the cases that they classify as "dementia traumatica" they describe the following lesions: "Small, old hemorrhagic foci, transformed into cysts or into glial scars, diffuse chronic lesions of the nervous elements, chronic diffuse lesions of the vessels," and they end the discussion by saying: "One must admit that following trauma there may be localized gross lesions or very mild diffuse lesions following which a chronic process of gliosis and degeneration of nerve cells takes place."

Of greatest importance, because they seem to be purely cases of concussion, are the five cases reported by Cassasa.[13] The patients were between the ages of 12 and 41. There was a history of injury of the head without laceration of the scalp. In two cases the early history was not ascertained, and the patients were admitted to the hospital in a semiconscious state. The other three patients were operated on, one with a preoperative diagnosis of cerebral edema, the other two of epidural hemorrhage. These three patients were momentarily unconscious at the time of the injury, regained consciousness for from three to twenty-four hours and then became unconscious again. The clinical observations following the concussion in all cases were irritability, increased deep reflexes, hypersensitiveness to light and sound, contracted but reacting pupils, and clear cerebrospinal fluid. These five cases were the only ones of the kind encountered in ten years during which many

thousands of necropsies in traumatic cases were performed by Schultz, Norris and Cassasa. There was no fracture of the skull. The meninges were intact. The cerebrospinal fluid was clear. Convolutions and sulci were normal and lacerations or contusions were not present. Gross section showed many scattered minute hemorrhages, a typical one of which is seen in figure 2. These were found throughout the entire brain, including the cerebellum. Microscopic examination showed areas of hemorrhage limited to the perivascular lymph spaces and others also within the immediately adjoining substance of the brain. In all these cases the patients died within a few days after the injury, and autopsies were performed within twenty-four hours after death. It is

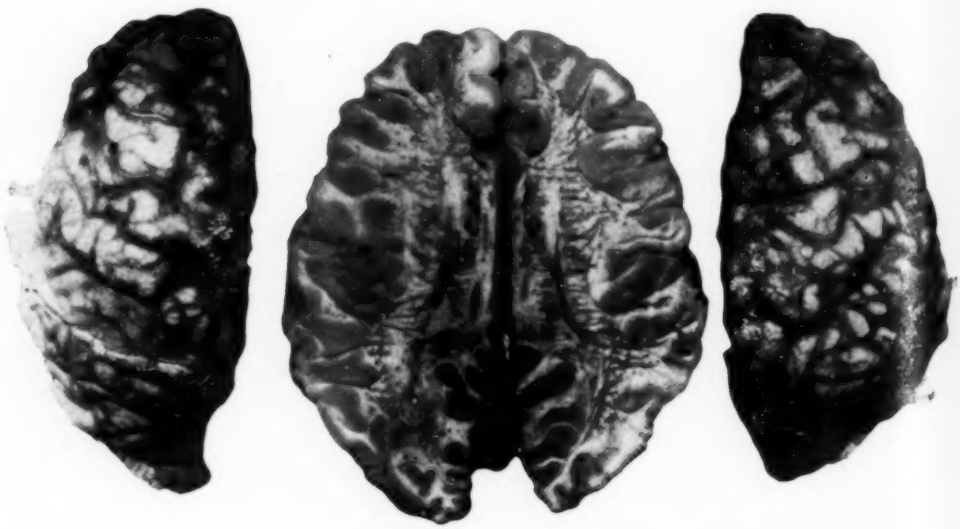

Fig. 2.—Typical scattered minute hemorrhages.

for this reason that there was not much neuroglial or other cellular reaction. However, the appearance of the section showing perivascular extravasation is strikingly similar to the perivascular reaction seen in epidemic encephalitis.

TRAUMATIC ENCEPHALITIS

The conception of traumatic encephalitis as a type of the encephalitides is not new, but reference to the literature will disclose that the pathologic condition is based on a study of injuries of the brain caused by projectiles or other forms of direct trauma, which also involve the skull and meninges and are followed by infection. The encephalitis that ensues is seldom purely traumatic, because the descriptions refer to "abscesses of greater or lesser extent, with more or less diffuse inflam-

matory reaction, manifesting itself in the perivascular spaces by the presence of leukocytes and accompanied at the edge of the involved area, by the appearance of numerous giant neuroglial cells. Necrotic lesions coexist with hemorrhagic ones producing serious involvement of the axis cylinders. In the neighborhood of the loss of substance, necrotic lesions are generally extensive or may be widely disseminated." Foix,[16] from whose paper this description is taken, shows a picture of

Fig. 3.—Hemorrhage into perivascular region and surrounding brain tissue.

this type of reaction and in the footnote accompanying the figure,[22] which was borrowed from Bouttier, he stresses the absence of inflammatory elements in the perivascular spaces in the noninvolved areas and calls attention to the often sharp line of demarcation between the necrotic areas of the brain and adjacent normal cortex. This type of traumatic encephalitis is well known. Unfortunately, however, it is of only little

22. Footnote 16, figure 9, p. 146.

interest in connection with the subject of pure concussion. The photograph of the sections from the brain in Cassasa's series,[13] which we have reproduced through his kindness, and the photographed sections of the brain that we studied show definitely the diffuse perivascular hemorrhagic infiltrations that are so strikingly absent in the illustration used by Foix. The similarity of these hemorrhagic extravasations in the perivascular and adjoining tissues to the perivascular infiltrations seen in early cases of acute encephalitis is striking. Figure 5, taken

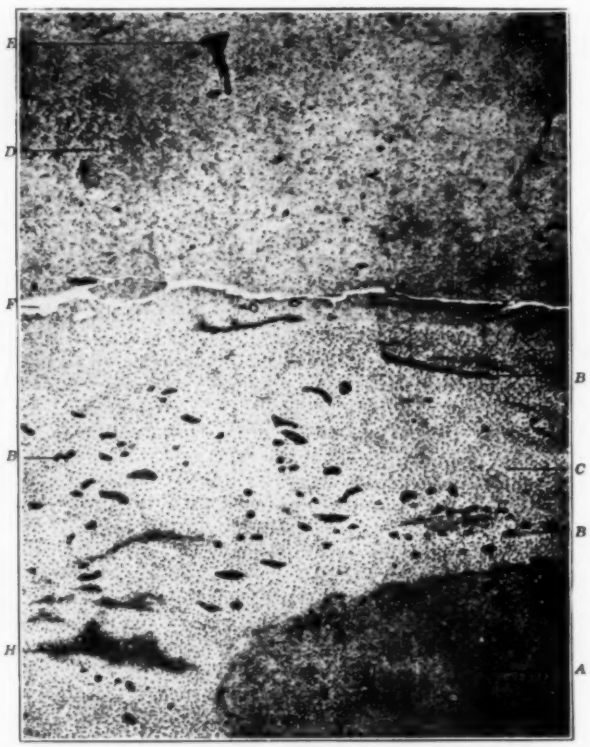

Fig. 4.—Necrotic hemorrhagic and normal brain tissue in same section ((illustration used by Foix[16] borrowed from Bouttier's Etude des traumatismes cérébraux récents, Thèse de Paris, 1918). *A*, indicates island of necrosed cerebral substance; *B*, newly formed vessels; *C*, granular cells mixed with products of disintegration; *D*, normal cerebral substance adjoining necrosed area. The absence of inflammatory infiltration and hemorrhages should be noted. *E*, blood vessels bordering normal cerebral substance. The absence of inflammatory elements in perivascular space should be noted. *F*, cleft of separation between diseased and healthy tissues. This is caused by retraction following the process. *H*, hemorrhagic zone with abundant granules of ferruginous pigment.

from a series of sections in a case of encephalitis, is reproduced for comparison.

The lesions in concussion of the brain and in epidemic encephalitis are, of course, different in certain important particulars. There is, notably, the absence of any considerable neuroglia reaction, and nothing of a really definite nature is seen in the other cellular elements of the nervous system. As would be expected, the cases of concussion that

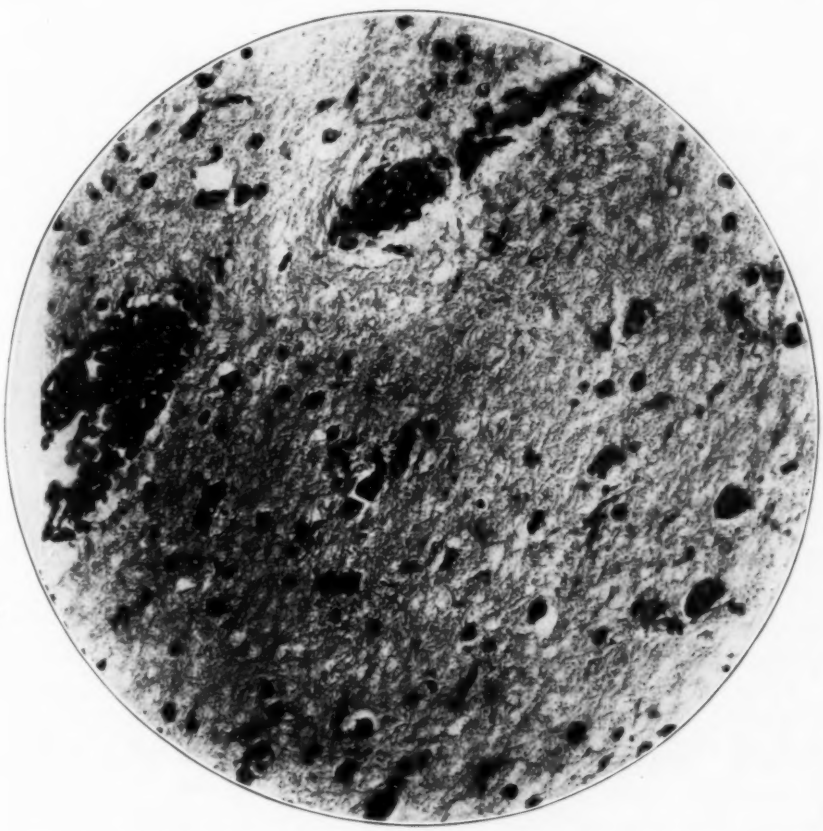

Fig. 5.—One of a series of sections taken in a case of encephalitis (photographed through the courtesy of Drs. Tilney and Howe).

come immediately to autopsy show chiefly the hemorrhagic and acute thrombotic picture. Usually the disturbed blood vessel is in the periphery, the white or gray substance of the deeper parts of the brain being comparatively free from injury. The pia-arachnoid suffers most. These points are illustrated by our case and confirm the remarks of

Greenacre [23] concerning fifteen selected cases of traumatic pontile hemorrhage, twelve of which were associated with basal skull fracture of the posterior and middle fossae. His series showed the hemorrhages as almost uniformly punctate, frequently grouped around the periphery of the pons, and connected with small subpial extravasations and numerous lacerations. The later glial proliferation and the various stages of sclerosis of the ganglion cells, together with evidence of degeneration in the axis cylinders, dendrons and the fibrous and cellular process of glial scar replacement probably follow as a result of what Meyer refers to as a typical wallerian degeneration. These secondary degenerative reactions probably occur rarely indeed. Surely in the vast majority of cases of concussion, recovery with almost complete resolution of the diffuse hemorrhagic process takes place, even granting that this occurs to a varying degree in every case of concussion of the brain. It has not been realized, perhaps, how many people in the general population have suffered concussion without, however, so far as one is able to judge clinically, showing any lasting effects. Taking into consideration the number of children who have had falls with resultant injury to the head and varying degrees of the concussion syndrome, and the great number of boys who have concussion in consequence of injuries while at play, and including also those who in adult life fall from horses, suffer automobile accidents and industrial injuries of various kinds, one can form a picture of the enormous number of persons in the general population who have, at some time or other, suffered a concussion of the brain. Nevertheless, permanent disabling clinical phenomena following concussion of the brain, with or without fracture of the skull, are not commonly encountered. This is in line with our experiences with craniocerebral war wounds. The number of cases of traumatic insanity or epilepsy, or of cases of tumor of the brain which develop following such injuries, is surprisingly small. Nevertheless, when the hemorrhagic process has been so marked and so diffusely present as in the brain we studied and in Cassasa's case,[13] it cannot be otherwise than that certain mental and somatic disabilities, chiefly the former, will follow, developing with the secondary diffuse glial and ganglion-cell degenerations. The degree of this diffuse secondary process determines the nature and degree of the clinical phenomena, with one important reservation. Sooner or later, certain instinctive or environmental influences operate to produce a group of emotional symptoms which it is difficult not to classify as purely psychogenic in origin. A discussion of these factors has no place in this paper. Our opinion of the psychopathogenesis of many of these purely psychogenic reactions, which are

23. Greenacre: Bull. Johns Hopkins Hosp. **28**:86 (Feb.) 1917.

classified as "industrial neuroses," has been discussed elsewhere.[24] It is helpful to recall in this connection that purely psychogenic symptoms undoubtedly occur in epidemic encephalitis or in any other organic disease of the central nervous system and that emotional situations can, and often do, add a psychogenic coloring to the picture.

REPORT OF A CASE OF CONCUSSION OF THE BRAIN

History.—The patient, who was in the early thirties, was injured while riding on a motorcycle. He was caught between a passenger automobile and a truck, was thrown to the pavement and rendered immediately unconscious. He was taken to the Polyclinic Hospital, where he soon regained consciousness. Later in the day a diagnosis of meningeal hemorrhage was made, and the patient was prepared for operation. The skull was trephined, but the meninges were not opened. The patient began to show disturbance of respiration and heart action while on the operating table. The operation was suspended, and the man died soon afterward (within thirty-six hours of the accident).

Postmortem Examination.—Necropsy was performed by Dr. Cassasa, through whose courtesy the brain was obtained, within twenty-four hours after the patient's death. The brain was placed in a 10 per cent mixture of commercial solution of formaldehyde. The skull, examined carefully, did not reveal any evidence of fracture. There was no fracture of the long bones.

The pathologic examination was made possible through the courtesy of Dr. Leon H. Cornwall, in whose laboratory the sections were prepared and examined. Dr. Cornwall's description follows.

Pathologic Examination.—The brain weighed 1,400 Gm. The conformation was normal. The leptomeninges covering the whole brain, more especially over the convexity of the cerebral hemispheres, were milky and edematous, and there was moderate distention of the pial veins. There were small petechial hemorrhages scattered through the centrum ovale, corpus callosum, and pons. These were especially marked in the corpus callosum.

On microscopic examination of the hemorrhagic areas, some of the red blood cells were basophilic in reaction, having an affinity for the hematoxylin stain. Sections from all parts of the brain, except the cerebellum, revealed a vacuolated appearance. The tissue of the brain appeared spongy or honeycombed. Large clear spaces were present around the blood vessels, ganglion cells and glia cells. In some instances two or three glia cells were in the center of a clear space. The ganglion cells were elongated and appeared compressed.

One is always loath to attribute too much importance to a vacuolated appearance such as was seen in this case because of the possibility of its being due to artefact. The fixation had been accomplished by means of 10 per cent commercial solution of formaldehyde. I am informed by Dr. Cassasa that a similar condition has been encountered in only five brains among the large number that he has examined during the past five years. All of these were from cases of death from concussion of the brain. The moth-eaten appearance of this brain is unusual in my experience with brains fixed in solution of formaldehyde, which includes the examination of speciments that have been preserved as long

24. Osnato, Michael: Industrial Neuroses, Am. J. Psychiat. **5**:117 (July) 1925.

as fifteen years. The rarity of this condition, therefore, and the diffuse distribution throughout the brain inclines me to attribute pathologic significance to it.

A second feature of interest was the presence of fibrin thrombi in the vessels of the meninges, choroid plexuses and cortical vessels. Such thrombi are frequently encountered as postmortem changes in the cavities of the heart and in the branches of the aorta and pulmonary vessels. Such agonal thrombi, however, do not completely fill the lumens of the vessels in which they are situated. In this case the thrombi completely filled many of the vessels and were intimately attached to the intima. Another unusual feature was the basophilic staining reaction given by the thrombi. Ordinarily, fibrin stains with eosin and not with hematoxylin. A similar tinctorial reaction was noted in the walls of some of the smaller blood vessels. The staining reaction observed was similar to that given by lime salts, and we have deduced the tentative

Fig. 6.—Transverse section of the brain, showing hemorrhages in the periphery of the pons and corpus callosum.

conclusion that as a result of the commotion there was disturbance in the colloidal equilibrium of the calcium in the blood stream facilitating its precipitation.

Many fat emboli were encountered in the small capillaries of the brain. These were demonstrated in sections treated by the Marchi method and embedded in celloidin, and also in frozen sections stained by the Herxheimer technic. In the absence of fractures, in this case it would seem reasonable to assume that the trauma to the medullary substance of the bones was sufficient to have produced this response, which is ordinarily associated with fractures. With the Mallory stain, the red blood cells, both intravascular and extravascular, were metachromatic in staining reaction, some being stained orange and others a bluish tint. Many of the clear spaces in the tissue stained a delicate blue with the Mallory stain.

The Purkinje cells of the cerebellum showed a great variability in their reaction to the Nissl stain. In the same field some were chromophilic, others chromophobic, and others achromatic. This observation was constant throughout the cerebellum. Hemorrhages were not encountered in the cerebellum, nor was the moth-eaten appearance noted.

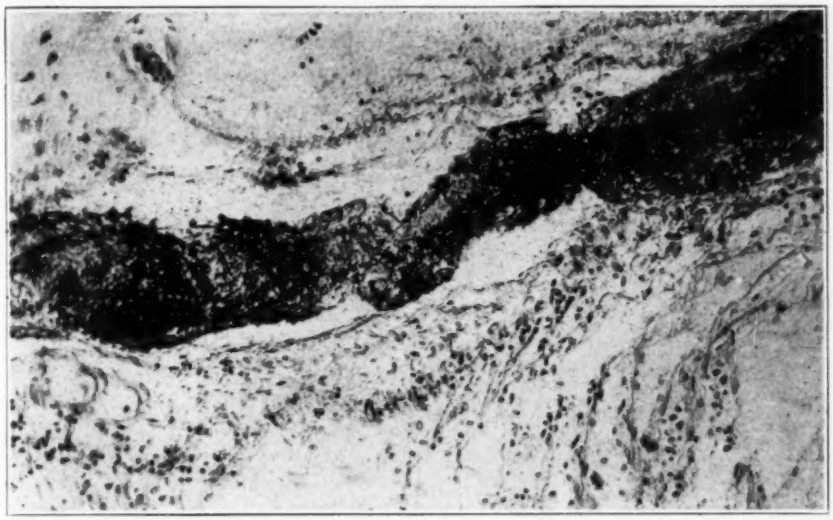

Fig. 7.—Moderate edema and congestion of pia. Fibrin thrombus in pial vessel.

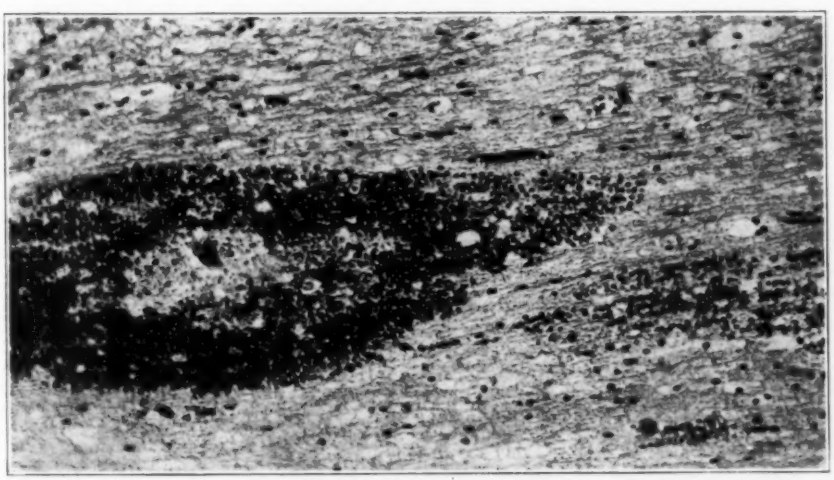

Fig. 8.—Tip of left frontal lobe. Low power. Hematoxylin and eosin stain, showing hemorrhagic extravasation.

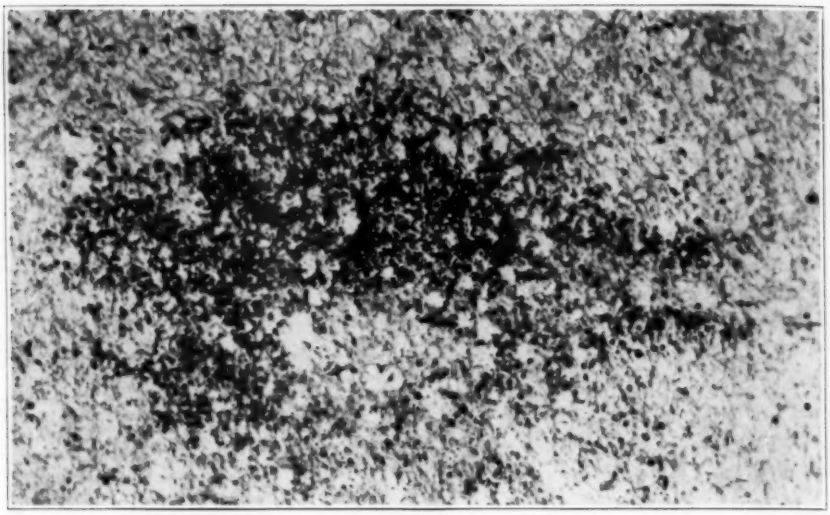

Fig. 9.—Hemorrhage in corpus callosum. Low power. Hematoxylin and eosin stain.

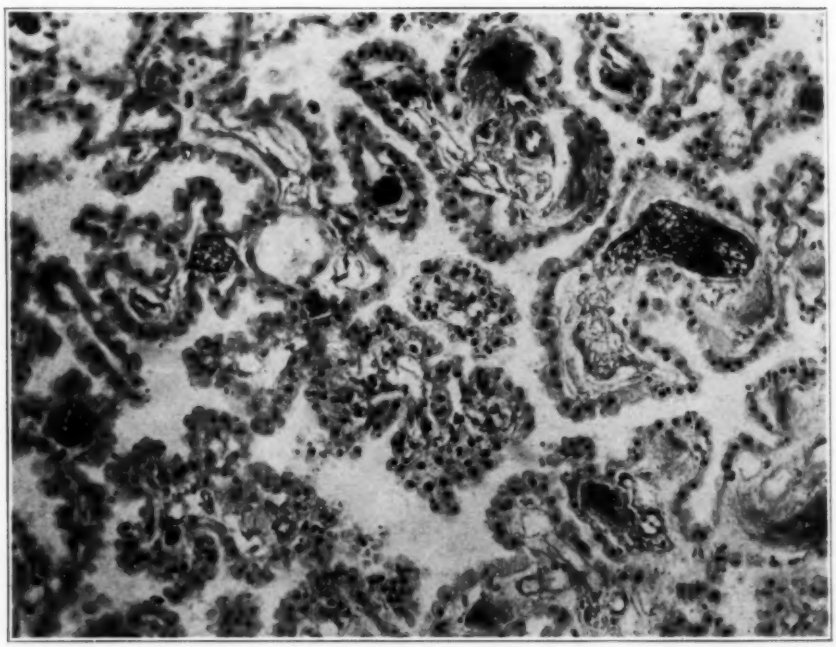

Fig. 10.—Fibrin thrombus—choroid plexus. Lower power. Hematoxylin and eosin stain.

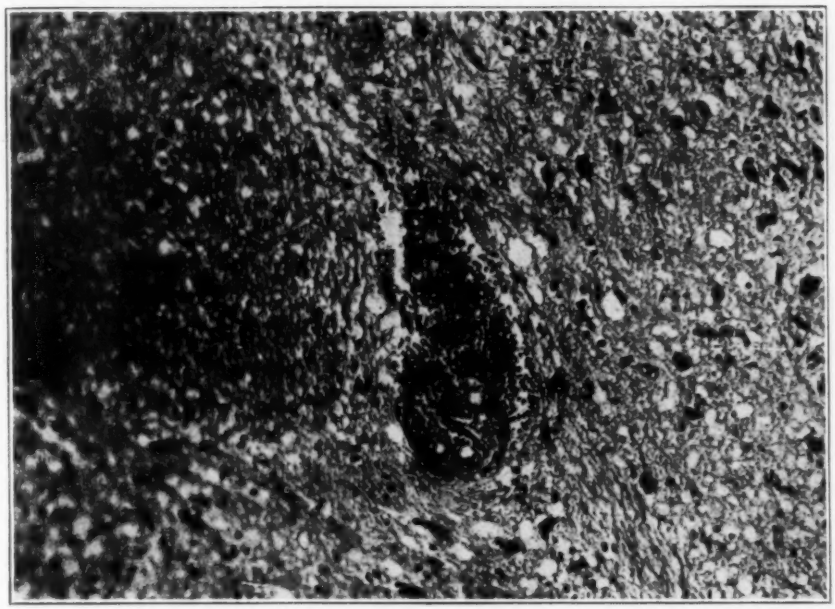

Fig. 11.—Hemorrhage in pons. Lower power. Hematoxylin and eosin stain.

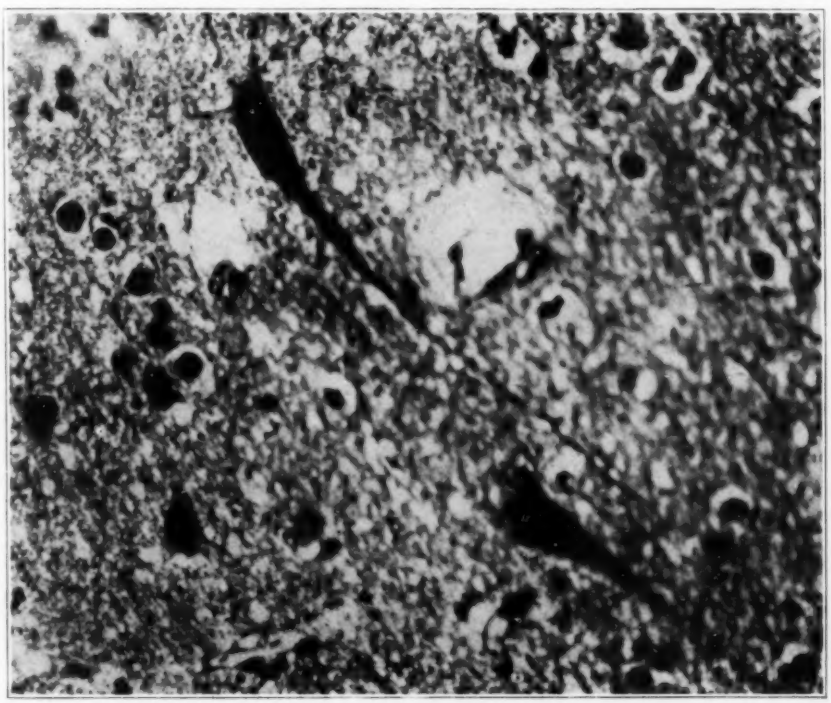

Fig. 12.—Uneven staining of pyramidal cells and vacuolated appearance of cortex. High power hematoxylin and eosin.

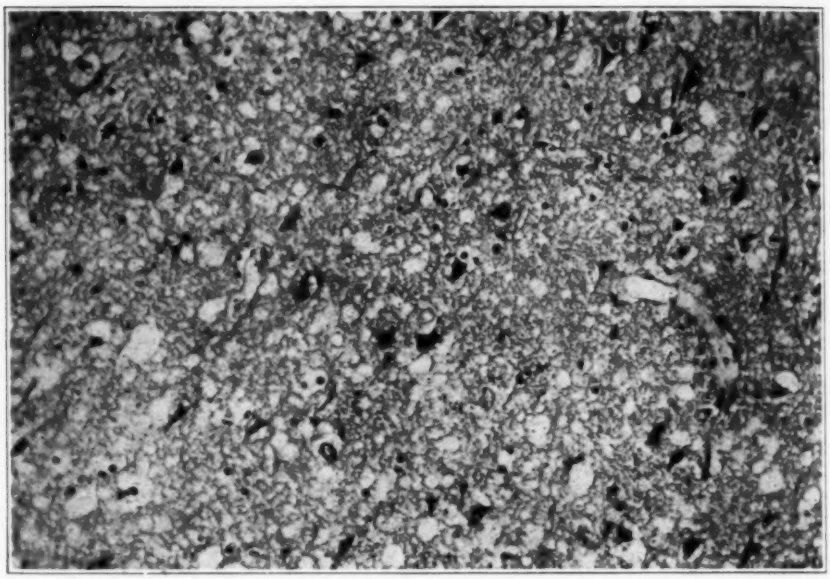

Fig. 13.—Vacuolated appearance in cortex. Low power. Hematoxylin and eosin stain.

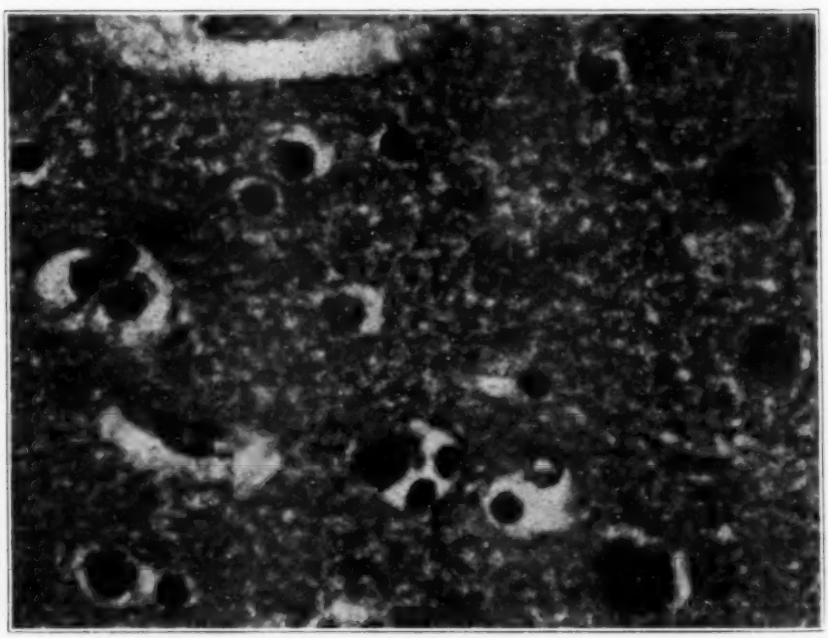

Fig. 14.—Vacuolated appearance in deeper layers of cortex. High power. Hematoxylin and eosin stain.

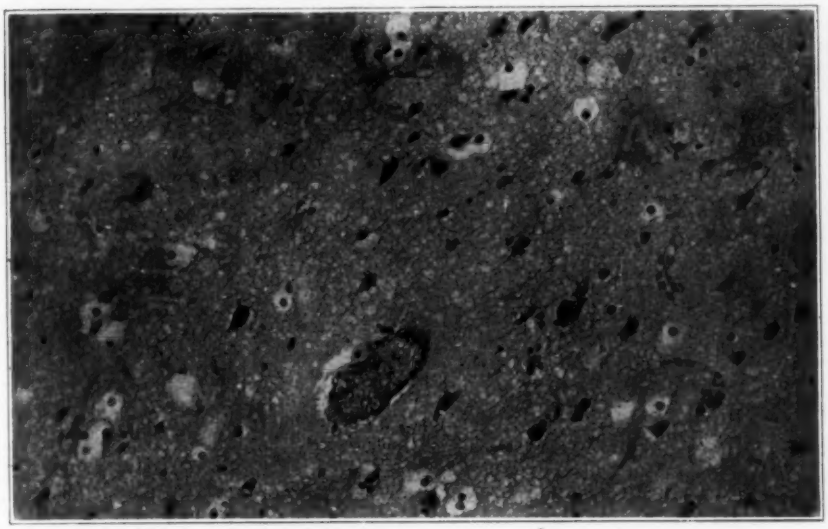

Fig. 15.—Tip of frontal lobe, showing état criblé and fibrin thrombus. Low power. Hematoxylin and eosin stain.

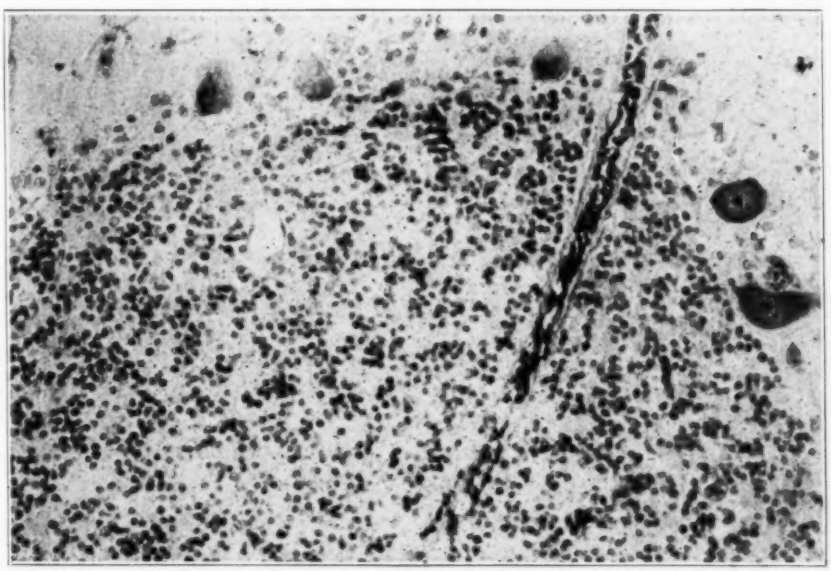

Fig. 16.—Uneven staining of Purkinje's cells and chromatolysis in cerebellum. High power. Hematoxylin and eosin stain.

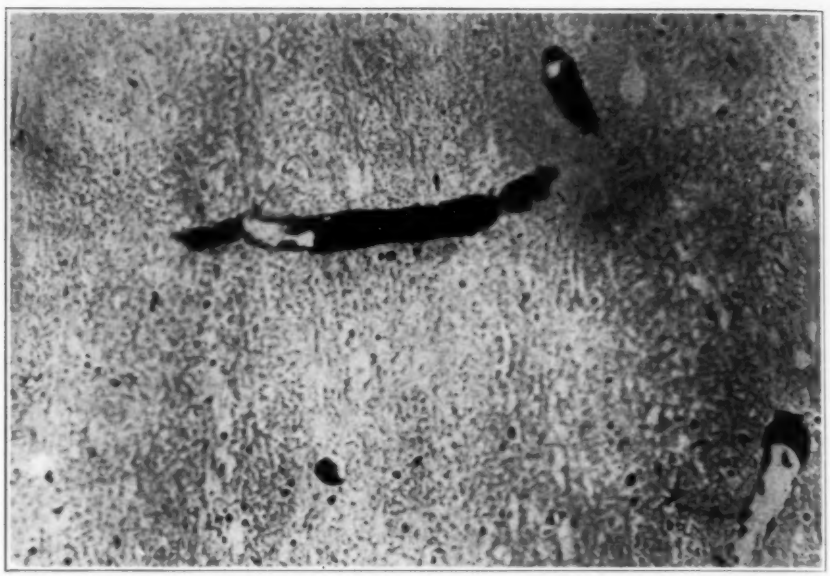

Fig. 17.—Concussion, showing fat emboli.

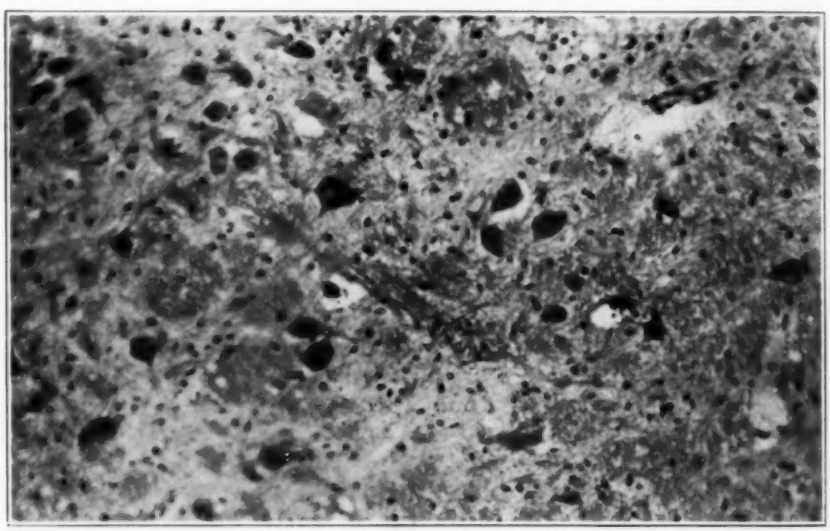

Fig. 18.—Medulla — nucleus ambiguus. Low power, showing chromatolysis and uneven staining of cells.

Comment.—The slides showing cellular changes are reminiscent of Mott's material [11] and recall his assumption that the changes in the nuclei of the medulla probably explained the deaths in his cases of commotio. It seems hazardous to say definitely that the cellular changes in the medullary nuclei in our case were sufficiently severe to explain the death. That there was marked disturbance of respiratory and heart

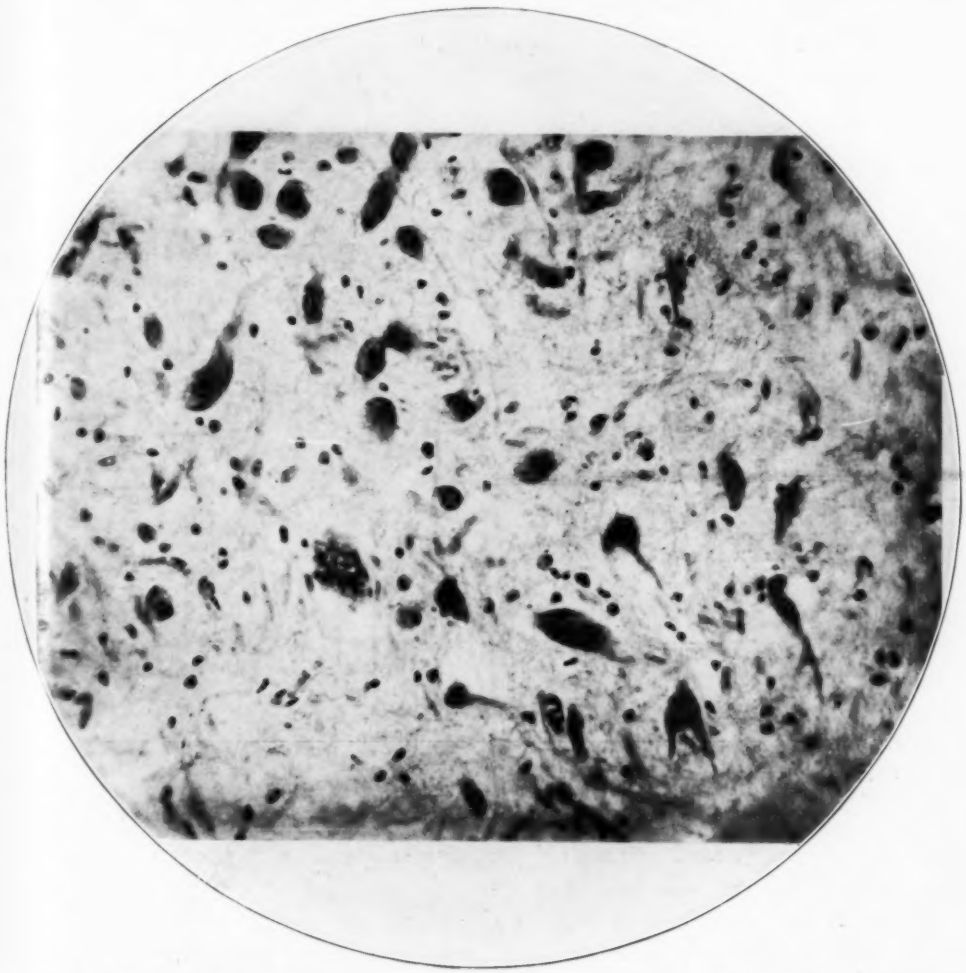

Fig. 19.—Nucleus ambiguus. Chromatolysis and fibrin thrombus. Weigert's iron hematoxylin stain. × 700.

rhythm due to poorly functioning centers in the medulla, however, may be assumed from a consideration of the patient's behavior immediately after the anesthesia was begun. It is conceivable that respiratory nuclei in the damaged medulla may have had placed on them a strain for which they were entirely inadequate.

STATISTICAL STUDY OF SUBJECTIVE SYMPTOMS IN THE POST-CONCUSSION CASES AND EPIDEMIC ENCEPHALITIS

Certain special factors must be eliminated from discussion in considering a comparison of the symptoms. For instance, the symptom of pain at the site of injury would have no basis for comparison in the cases of epidemic encephalitis.

Headache.—The symptom of headache localized at the point of injury to the skull is often annoying. Trotter's [25] attitude toward surgical treatment in these cases is surprisingly radical. He would have us

TABLE 2.—*Symptoms Present in Concussion and Encephalitis*

		Concussion 100	Encephalitis 100
Dizziness		51%	22%
Giddiness		6%	5%
Tinnitus		19%	....
Visual disturbances	Dimness	....	8%
	Blurred	16%	13%
	Double	....	54%
	Scotoma subjective	3%	1%
	Optic atrophy	2%	1%
Headache		69%	68%
Pain in eyeballs		7%	6%
Nervous fears		11%	12%
Drowsiness		8%	33%
Hypersensitiveness, especially to noises		16%	7%
Delirium		11%	39%
Restlessness		22%	25%
Depression		8%	7%
Disturbance of sleep		38% sleeplessness at night	51% somnolence 32% sleeplessness at night
Irritability and moodiness		23%	14%
Emotionalism		10%	18%
Fatiguability		9%	13%
General weakness		21%	44%
Convulsion		1%	2%
Palpitation		1%	1%
Stuttering and stammering		3%	2% aggravation of pre-existing disturbance
Nausea		12%	6%
Vomiting		6%	8%

believe that the complaint of headache at the site of an injury to the head, in the absence of any objective signs of injury to the brain or of evidence of generally increased intracranial pressure, is enough, if the headache is persistent and made worse by stooping or bending, to warrant decompression with opening of the dura over the exact point of impact. When one considers that Trotter applies the same type of reasoning even to ordinary contusions of the scalp, in which there is no question of concussion, the radical nature of his recommendations will be more apparent.

In table 2 it will be seen that headache is present in almost as many cases of encephalitis as in those of postconcussion neurosis. It would

25. Trotter: in footnotes 1 and 3.

appear almost as good reasoning to suggest an osteoplastic flap or a subtemporal decompression in the cases of encephalitis as in the cases of concussion.

Visual Disturbances.—An interesting situation is encountered when one analyzes the visual disturbances in the two conditions. The special symptom of diplopia is explained by the location of the pathologic process in epidemic encephalitis, but otherwise the symptoms referable to vision are almost as frequently encountered in one condition as in the other (table 2). Optic atrophy was encountered in 2 per cent of the cases of concussion and in 1 per cent of the cases of encephalitis. In both of the postconcussion cases, the optic atrophy resulted from retrobulbar neuritis, due to fracture of the anterior fossa involving the orbital plate of the frontal bone or the lesser wing of the sphenoid.

It seems certain that the visual disturbances complained of in the postconcussion cases have just as positive an organic basis as in the cases of encephalitis. Unfortunately, many of our patients were seen months, sometimes even years, after the accident and these objective phenomena were no longer demonstrable. Ah excellent piece of work on the recovery of the fields of vision in concussion injuries of the occipital cortex by Hine [10] is worth reading in this connection. According to Hine, even cases "of definitely gross injury to the brain substance may clear up so completely that they are indiscoverable later on, or discoverable only by very careful examination." These observations confirm the more extensive ones made by Lister and Holmes.[26] They probably explain the so-called subjective visual disturbances of blurred vision, scotoma, difficulty in concentration when reading and so many other types of visual complaints which on examination are not confirmed by objective observations. The conclusions of Hine may be briefly summarized as follows: (1) There may be complete hemianopia. In civil practice, vascular lesions in this region are usually characterized by escape of the macular area, because of overlapping of the arterial supply of the posterior and middle cerebral vessels. (2) The visual fields are gradually restored from center to periphery. This is true also of scotoma. (3) The field of vision returns first in the upper quadrant, because most of the injuries to the occipital lobe have been above the occipital pole. Therefore, the occipital cortex above the calcarine fissure, which represents the lower part of the field of vision, bears the brunt of the blow. (4) Color perception follows after the return of perception of white. Finally, the appreciation of colors becomes more and more acute, so that small colored objects can be distinguished further and further away. (5) It is important, when investigating injuries of the occipital lobe, to use small colored test objects as well as white ones.

26. Lister and Holmes, G. M.: Proc. Roy. Soc. Med., 1916, vol. 9.

because a partially recovered lesion may be missed and the fields found to be full. In this connection, Hine states: "Even a gross injury of the cerebral substance may clear up and leave no trace behind. If this fact is borne in mind, I believe that quite a number of overlooked lesser injuries to the cerebral cortex of the occipital lobe—which could be classed as concussion—will be discovered." These observations of Hines

TABLE 3.—*Oculomotor and Pupillary Disturbances in Concussion*

Areus Senilis	Artificial Dilatation	Oculomotor Weakness	Subjective: Blurred Vision	Subjective: Scotoma	Anisocoria	Contour of Pupils	Defective Vision	Fundi
	Yes	Convergence weakness	Yes	....	....	....	....	
	Yes	Convergence weakness	....	....	....	....	....	
		.....	....	....	....	....	Yes	Arteriosclerotic
		.....	....	....	....	....	....	Arteriosclerotic
		.....	....	....	Yes	....	....	
		Right internal rectus	....	....	Yes	Irreg.	Yes, cataract	
		Convergence weakness	Yes	Yes	....	....	....	
		Convergence weakness	....	....	....	....	Yes	
		Right external rectus	....	....	....	....	....	
		Right external rectus	....	....	....	....	....	
		.....	....	....	....	....	Yes	Arteriosclerotic
Yes	....	Convergence weakness	....	....	....	....	....	Arteriosclerotic
		.....	....	....	Yes	Irreg.	....	
		.....	....	....	....	....	Yes	Right secondary optic atrophy, left primary optic atrophy
		Left internal rectus on convergence	Yes	....	Yes	....	....	
		.....	....	....	Yes	....	....	L. old choroiditis
Yes	....	.....	....	....	Yes	Irreg.	Yes	Arteriosclerotic
		.....	....	....	....	....	....	Arteriosclerotic
	Yes	Right internal rectus on convergence	....	....	....	....	....	
		Convergence weakness	Yes	Yes	....	....	Yes	
		R. internal strabismus	....	....	....	....	....	
		Right internal rectus, weakness on convergence; right external rectus on fixation to right	Yes	....	....	....	Yes	Right optic atrophy
		Right internal rectus on convergence	....	....	....	....	....	
		.....	....	....	....	....	Yes	
		Convergence weakness	....	....	....	....	....	Arteriosclerotic
		.....	....	....	....	....	....	Arteriosclerotic
		.....	....	....	....	....	....	Arteriosclerotic
		Left internal rectus on convergence						
		Convergence weakness	....	....	....	....	....	Arteriosclerotic
		.....	....	....	....	....	....	Arteriosclerotic
		.....	Yes	Yes	Yes	....	....	
		Convergence weakness	....	....	....	....	....	Arteriosclerotic
		.....	....	....	....	....	....	Arteriosclerotic
		Left internal rectus, ext. strabismus	....	....	....	....	....	Arteriosclerotic
		Right internal rectus on convergence	....	....	Yes	....	....	Arteriosclerotic
		Convergence weakness	....	....	....	....	....	Arteriosclerotic

are all the more important because, with the exception of one case which was used to illustrate a special point, namely, the importance of careful examination of recovering cortical lesions, all the cases were examples of simple concussion, without fracture of the skull and with simple wounds of the scalp as the only objective external evidence of injury to the head. Hine repeatedly mentions the fact that within a few weeks patients recover completely from even absolute hemianopias, as, for

example, in his case 4. It is therefore readily understood why, in our cases, the tests for visual acuity and fields were regularly negative, even in the presence of complaints regarding vision.

Hearing.—A point that requires some explanation is the fact that 19 per cent of the patients with concussion complained of tinnitus, whereas none of the patients who had encephalitis did so. The symptom of tinnitus can be best appraised if it is taken in connection with the complaint of impaired hearing. In 32 per cent of the cases of concussion this complaint was made; it was not made in the cases of encephalitis. Here again, it is a question of the site of the lesion more than a difference in its nature. It is notorious how frequently the

TABLE 4.—*Impaired Hearing*

Impacted Cerumen	Ruptured Drum	Malingering	Retracted Drums	Vestibular Disturbance	No Vestibular Disturbance
5	2	5	20	5	7

TABLE 5.—*Impaired Memory-Complaint*

Cerebral Arteriosclerosis	Age		Fractured Skull	
No	32	Memory good.....	No	
Yes	45	.....	No	
Yes	53	.....	No	
No	43	Tendency to become confused, is retarded, no memory defect, retention and recall	Yes	
No	36	Posteoncussion confusion, very poor attention, recent memory poor	Yes	
Yes	46	.....	Yes	
Yes	59	.....	Yes	
Yes	56	.....	Yes	Not confirmed by roentgen ray
No	47	Memory good.....	No	Hysterical amnesia
Yes	55	.....	No	

mechanisms of the middle and internal ear are disturbed with injuries of the skull, and it is equally well recognized that the eighth pair of nerves is not commonly disturbed in cases of epidemic encephalitis. Table 4 illustrates the fact that, in concussion cases without fracture, disturbances in hearing which persist are usually due to other conditions. In twelve cases of defective hearing caused by fracture of the base, five patients had vestibular disturbances also.

Memory.—The question of impaired memory is of interest. From table 5 it will be noted that even in the ten cases in which this complaint was made, it was confirmed by examination in only eight, and in one case there was a definite tendency for the patient to become confused, which undoubtedly accounted for the complaint; yet on examination, no loss of retention or recall of recent or remote memories could be demonstrated. In another case of a patient, aged 36, without clinical evidence

of cerebral arteriosclerosis, there was confusion, poor attention and only a disturbance of recent memory with complete amnesia for the injury and part of the hospitalization period. It is interesting to note that this patient had suffered a fracture of the skull, and in addition had a long period of unconsciousness, with delirium which lasted for several days. Obviously, this was a case in which there was a good deal more than simple concussion. In six cases, which make up the remainder of this 10 per cent, the memory defect was definitely similar to that seen in cerebral arteriosclerosis; it was patchy, with fairly good recent memory, hazy data when attempts to recall were made, and a tendency to become tired, irritable and confused if the examination was prolonged. In other words, a study of table 6 shows that in only two cases was there any memory defect that could not be explained by the presence of cerebral arteriosclerosis, and that in both these cases there was definite evidence of a fracture of the skull, an injury likely to cause a good deal more cortical damage than probably occurs in cases of simple concussion. It seems certain, therefore, from the study of our material that an organic type of memory defect is not found in the postconcussion neuroses. Regularly, however, mild confusions with a tendency to tire easily on insistent, persistent questioning, and a characteristic delay in the responses, especially if the patient is seen early, are the features of the mental side of these cases. One would expect, after recalling the complete functional recovery experienced from severe visual defects due to cortical injuries, that rapid recovery takes place in memory defects in simple concussion. Accompanying the tendency to confusion and the retarded responses in the early cases of concussion, there is apt to be a mild depression which perhaps is responsible for the slow, rather sad-toned, monotonous type of speech. The patients in late cases do not show this feature. On the contrary, some of these may be irritable, moody, easily excitable and talkative. But many, perhaps most, are depressed, with disinclination to talk and great irritability. These disturbances in speech are entirely different from the type seen in epidemic encephalitis. Here again, the location of the lesions unquestionably explains the difference.

Disturbance of Sleep.—In the comparative table 2 are noted disturbances of sleep that are often strikingly similar in the two conditions. In the first weeks of the concussion phenomena, drowsiness, even somnolence, is the rule, but thereafter (in 38 per cent of our cases) sleeplessness, at night especially, is often the bitterest complaint. The type of disturbance of sleep encountered in epidemic encephalitis is frequently an inversion of the sleep phenomenon. These patients are able to sleep or at least drowse during the day, but are restless at night, particularly in the subacute and chronic stages of the disease. Fifty-one per cent of the patients with epidemic encephalitis suffered from somnolence in the

early months of the disease and were drowsy a good part of the time. Thirty-two per cent of the patients, representing chiefly subacute or chronic cases that have lasted for a year or more, often complained bitterly about inability to sleep or rest at night, with drowsiness in the daytime.

Dizziness.—A point regarding the headache in all of these cases of concussion may be mentioned again: that it is almost invariably accompanied by the complaint of dizziness. If one combines the complaints of dizziness with the rather indefinite complaint of giddiness, one finds that it is present in fifty-seven of the 100 cases, whereas headache is present in 69 per cent. We have learned to confine the term dizziness to those cases in which an actual vertigo is present, confirmed in a great many cases by disturbances of station or gait, the presence of nystagmus on changes of posture, or some disturbance of the after turning Bárány reactions. The term giddiness should, in our opinion, be limited to those complaints of peculiar subjective feelings of unsteadiness connected with subjective visual disturbances, such as a sensation of blurred perception, of moving objects, or of clouding of vision. Probably the six cases of giddiness included in this tabulation really belong with various types of visual disturbances enumerated, rather than in the group showing dizziness.

Other Features.—It is interesting to note how nearly equal the figures are for pains in the eyeballs in both conditions under discussion and also for the subjective complaint of nervous fears. The latter symptom covers the whole field of phobias and need not be gone into in detail here; suffice it to say that it may be disturbing, and that in several of the cases studied it was practically the only reason for disability at the time of examination. Whether emotional factors other than the injury itself are responsible for these phobias in the two conditions of such definitely organic origin is something that would not be profitable, perhaps, to discuss in this paper.

Blood Pressure.—The blood pressure readings in our cases of concussion were generally within normal limits. Table 6 shows in detail the cases that gave unusual readings. It can be seen that in nearly every case in which the blood pressure varied from the normal limits, a condition of the myocardium or of the cardiovascular system explained the symptoms. In one case the systolic blood pressure was 88, diastolic 56, with a tendency to slowing of the pulse. The patient was a girl, aged 18, who gave general clinical evidence of incipient pulmonary tuberculosis. In a patient, aged 35, seen only once, definite evidence of myocarditis was not apparent, and there was nothing unusual in the fundal or peripheral blood vessels that might be indicative of arteriosclerosis. A blood chemistry examination was not made in this case,

but clinically there was a suspicion that the patient might be suffering from nephritis. It would appear from our studies, therefore, that patients who have suffered a concussion of the brain and are seen some weeks or months after the injury do not present abnormalities of arterial tension in the absence of definite cause in the cardiovascular or renal systems that cannot be explained by the presence of some other condition.

THE OBJECTIVE SYMPTOMS OF CONCUSSION

Weakness of convergence was found in 22 per cent of the cases (table 3). In six of these there was demonstrable weakness of one of the recti. All of these patients had normal vision, but whether there were congenital or acquired disturbances of a refractive nature was not determined. A weakness of convergence occurs so commonly in normal adults (presbyopia) that it would be purely speculative to say that all

TABLE 6.—*Blood Pressure*

Pressure	Age	Myocardium	Arteriosclerosis
142/ 76	50	Chronic myocarditis, auricular fibrillation....	Marked cerebral and systemic
104/ 60	40	Not examined	No
88/ 56	18	In sixth interspace, tendency to bradycardia.	No; pulmonary tuberculosis ?
186/120	43	Cardiorenal disease	Marked cerebral and systemic
188/104	49	Heart enlarged to left, aortic second sound accentuated	Marked cerebral and systemic
138/ 76 to 162/ 84*	50	Chronic myocarditis and endocarditis.....	Marked cerebral and systemic
180/ 92	59	Chronic myocarditis	Marked cerebral
160/ 96	35	Cardiorenal disease	Marked cerebral
150/ 88	40	Normal	Cerebral and systemic
158/ 90	50	Normal	Cerebral
152/ 88	55	Normal	Cerebral and systemic

* Both readings during one examination.

of the twenty-two patients suffered from this symptom as the result of residual paretic phenomena in the muscles of the eye due to injury of the third nerve group of nuclei or the centers for binocular movements in the corpora quadrigemina or elsewhere. A residual weakness in one of the recti was, however, found in six cases and was probably responsible for the weakness of convergence. The patients who subjectively complained of blurred vision showed the abnormalities of visual acuity tabulated in table 3. There were eight patients with unequal pupils; three of these not only had a weakness of one of the recti of the same eye, but also had difficulty in convergence. Scotomas were complained of but were not confirmed objectively in three cases, and three of the patients had irregular pupils. The fields were normal in all these patients except the two who had optic atrophy.

The tremor in these cases was limited to the eyelids and the extended fingers. It was generally of moderate amplitude, irregular rhythm and moderate force and was made worse by physical or emotional stress. Attention should be called to the fact that the tremor in many cases of

epidemic encephalitis is not typical of the parkinsonian types occurring in middle or advanced age, secondary to arteriosclerosis or to senility. It is not true that all patients with parkinsonism from epidemic encephalitis have a tremor that exactly fits in with the rest of the parkinsonian picture. Many of them have a tremor only of the lids and of the extended hands of precisely the irregular type seen in cases of concussion.

The results of the Bárány examinations were taken up for investigation because of the importance attached to them by Eagleton [27] and others. Eagleton is positive that a syndrome of concussion of the brain or of increased intracranial pressure in the posterior fossa has been established. This he describes as reduction in the duration of nystagmus after stimulating the horizontal canals and complete absence or marked reduction of response from the vertical canals in the turning tests. With caloric tests, there is a reduced response on the affected side with prolonged interval between the application of the stimulus and the inception of the vertigo response. He also stated that "small, acute, and

TABLE 7.—*Tremors — Regular and Moderately Coarse of Medium Amplitude*

	Concussion of Brain	Epidemic Encephalitis
Eyelids	7	4
Hands	27	15
Paralysis agitans type	..	27

destructive lesions, such as hemorrhages involving the so-called 'vestibular pathways' will produce symptoms or give localizing manifestations. The outspoken vestibular manifestation of increased intracranial pressure —absence of reactibility of the vertical canals of both ears to the cold caloric—first described and properly interpreted by the author, while an early manifestation of increased intracranial pressure prevents an accurate localization by the vestibular reactions alone. It is of the greatest value, however, in the early diagnosis of increased intracranial pressure as it appears long before and independently of papilledema." Most of Eagleton's patients were seen shortly after the injury. Unfortunately, ours were seen months, and even longer, after the onset of the concussion. This may account for the difference in our observations. Our results are given in tables 8 and 9. It can be seen from them that the nature of the response is varied, yet there were in some cases definite indications of abnormality. In some cases the after-turning nystagmus, when the horizontal canals were tested, was only moderately reduced, but in others it was much more prolonged than is usual in normal persons. When the vertical canals were tested, the response either was absent as far as nystagmus went, which happened only infre-

27. Eagleton: Brain Abscess, p. 199.

quently as the table shows, or was prolonged, or was normal as to duration, but attended by excessive vertigo. In a number of cases the turning tests of the vertical canals could not be done because of the uncomfortable vertigo response. In one case the right ear (caloric test) showed

TABLE 8.—*Response to Vestibular Tests in Forty-Nine Cases of Fractured Skull*

	Cases	Abnormal Response	Normal Response	Not Tested
Fractured skull cases	49	14	5	30
Concussions without fracture	51	8	3	40
		Concussion with Fracture	Concussion without Fracture	
Abnormal vestibular responses	22	14	8	
Normal vestibular responses	9	6	3	

TABLE 9.—*Bárány Tests*

Fracture ?	Concussion ?	Nystagmus Duration after Chair and Caloric Test	Drum Perforated
No	Yes	H., 20 sec ; V., 50 sec.	No
No	Yes	H., 26 sec.; V., not done	Yes
No	Yes	H., 32 sec.; V., had to stop after 7 turns; was so upset he interfered with observation	No
No	Yes	H., 15 sec.; V., several seconds only	
No	Yes	H., 26 sec.; V., 25 sec., excessive vertigo response	No
Yes	Yes	?, turning made him very sick	Yes
Yes	Yes	H., 48 sec.; too sick to try V	No
Yes	Yes	H., 52 sec.; V., not tried	No
Yes	Yes	H., 19 sec.; V., 6 sec.	No
Yes	Yes	H., 32 sec.; douching right ear for 18 seconds induced nystagmus lasting 2 min. and 32 sec.	
Yes	Yes	Normal Normal	No
Yes	Yes	H., 16 sec.; too sick for V	No
Yes	Yes	H., 26 sec.; V., 24 sec.	No
Yes	Yes	H., 34 sec.; V., 24 sec.	No
Yes	Yes	H., 34 sec.; V., 24 sec.	No
Yes	Yes	H., 2 min.; marked vertigo	No
Yes	Yes	H., 38 sec.; V., not done	No
Yes	Yes	H., few sec.; V., few sec	No
Yes	Yes	Caloric tests: right ear, 3 min., no reaction; left ear, 110 sec., nystagmus 18 sec. and slight vertigo	No
Yes	Yes	H., 36 sec.; V., 19 sec.	No
Yes	Yes	H., 24 sec.; V., 24 sec.	No
No	Yes	H., 26 sec.; V., 25 sec.	No
No	Yes	H., 28 sec.; V., 26 sec.	No
Old fracture	Yes	H., 35 sec.; V., not done	No
No	Yes	H., 32 sec.; caloric, left ear vertigo in 70 sec., nystagmus lasted 2 min. 16 sec.	
Yes	Yes	Right, vertigo in 70 sec., nystagmus 1 min.; left, vertigo in 36 sec., marked nystagmus 3 min.	No
No	Yes	H., 26 sec.; V., not done	No
Yes	Yes	H., 64 sec.; marked vertigo	No
No	Yes	H., 44 sec.; V., 66 sec.	No
Yes	Yes	H., 38 sec.; V., 31 sec.	No
No	Yes	H., 26 sec.; V., 24 sec.	No
19 recent fractures 1 old fracture 11 without fracture	31 concussion	22 showed abnormal Bárány responses	2 perforated drums 29 not perforated

a dead vestibule; that is, there were no nystagmus or vertigo responses after douching with cold water at 60 degrees for three minutes. The left ear in this case had to be douched for 110 seconds before a slight vertigo was produced with a duration of nystagmus of only twelve seconds. This man had a fracture of the skull but apparently had given

no history of bleeding from the ears and did not have a perforated drum. This was one of those unusual cases of double traumatic hemorrhage into the internal ear. Many cases of undoubted concussion gave normal after-turning Bárány responses. Perhaps the most characteristic feature of the Bárány tests in these cases is a tendency to a prolongation of the after-turning nystagmus, but more particularly excessive vertigo. Occasionally, the vertigo was so severe and so obviously uncomfortable that the tests could not be completed. We were unable to make out any definite syndrome of concussion, based on the chair-turning and caloric tests, but dizziness on change of posture with nystagmus in any of the planes seems to us, even in the absence of definite Bárány responses, to point to injury to the vestibulocerebellar pathways. This is found not infrequently in early cases of concussion of the brain.

CONCLUSION

Anatomic and clinical investigations seem to show definitely that our conception of concussion of the brain must be modified. It is no longer possible to say that "concussion is an essentially transient state which does not comprise any evidence of structural cerebral injury." Not only is there actual cerebral injury in cases of concussion but in a few instances complete resolution does not occur, and there is a strong likelihood that secondary degenerative changes develop. When this happens, we have a condition which, clinically at least, resembles some of the reactions seen in encephalitis. We feel, therefore, that the postconcussion neuroses should properly be called cases of traumatic encephalitis.

DISCUSSION

DR. S. E. JELLIFFE, New York: Dr. Osnato's paper brings to my mind, as possibly of collateral interest, a situation that may arise in medicolegal circles. It has been my experience in the last year to be called, in two instances, into controversial relations with some of my confrères regarding the diagnosis and liability following concussion in which there developed, within from eight to ten days, an interesting syndrome, which from one point of view was regarded as the after-results of a concussion, and from another point of view was legitimately sustained as the result of an epidemic encephalitis.

One brief citation may be sufficient: An iron worker, of approximately 42, leaning underneath a heavy iron bar suddenly righted himself, without thinking, and hit himself with considerable force on the back of the head. He was rendered partially unconscious for a few moments and was taken home, but returned to his work more or less desultorily for five or six days. He then developed an influenzal, nasal situation, and in a hospital had a delirium lasting for about twenty-four hours, with diplopia and the beginnings of a distinct hemiplegic syndrome.

The hospital records were carefully studied, and it was legitimately sustained by one of the confrères in the court that this man had really had an epidemic encephalitis. About six months later, when I saw him, as one of the contestants in the situation, he had a beautiful hemiplegic syndrome, with a divergent squint and slight hemitremor of the hemiplegic side.

As my own experience medicolegally is slight (and yet I have seen two cases in the last year), it seems perhaps not inappropriate to speak of this type of situation which arises, in view of the epidemic extension of such disease and the possible temporal relationship of trauma.

Dr. T. K. Davis, New York: Neuropsychiatrists should recognize more clearly injuries of the head and should eliminate diagnoses of psychogenic factors when they are not there. They should avoid too great emphasis on vestibular tests because they are by no means on a scientific footing.

Dr. Ernest Sachs, St. Louis: Surgeons see these patients frequently. I have thought for a long time that the term "concussion" is most unfortunate, because clinically it is absolutely impossible to find a picture that is due to what might be called "concussion." Imperceptibly, it fades off into more severe states which have been proved to be the result of contusion and laceration of the brain.

It is my belief that all these conditions ought to be spoken of as contusions and lacerations, except perhaps the sort of case that Dr. Osnato referred to in quoting Trotter.

I have been much impressed with the fact that an important factor in these cases is the rapidly developing edema of the brain, and the method of treatment that has been used generally in the last five or six years as a result of the work of Weed demonstrates this.

Another point that I think should be emphasized, which surgeons have seen on a number of occasions, is that not infrequently there is a rupture of the subarachnoid space with a flow of cerebrospinal fluid into the subdural space, where it cannot be absorbed.

I believe that many of these cases of prolonged headaches may be caused by that factor. I do not know exactly how it could be demonstrated. I have seen cases in which the mere relief of the collection of fluid in the subdural space has cleared up the patients' symptoms immediately.

It seems that this organization would do a great service, especially as to the medicolegal aspect, if it would inaugurate a concerted movement to abolish the term "concussion" and to call all these cases "contusions and lacerations."

Dr. Julian M. Wolfsohn, San Francisco: While working with Colonel Mott in London during the war, I had the opportunity to see several hundred cases of commotio cerebri, and also many common hysterical shell-shock cases with cerebral symptoms.

Under Mott's direction, I studied the excitability of the labyrinthine apparatus by means of voltaic vertigo tests, using the galvanic current with the electrode applied to each mastoid region.

In the majority of the cases of true commotio, we were able to demonstrate a heightened irritability of one or both labyrinths. We were not able, due to press of work, to do the vestibular tests, but our results with these tests correspond to the results that Dr. Osnato has given.

Some of our cases of commotio cerebri came to autopsy, and Mott demonstrated marked punctate hemorrhages throughout the system, especially in the white matter of the cerebrum and the pons, etc.

The voltaic vertigo reaction was always within normal limits in the hysterical cases.

Voltaic vertigo is easily performed as the apparatus can be brought to the bed-side, and aside from the vertigo produced by the test, the patient is undisturbed.

DR. CHARLES E. DOWMAN, Atlanta: Those of us who are interested in neurosurgery feel that the habit of referring to or thinking of cases of injury of the head in terms of fracture of the skull is a great mistake. We are attempting to look on cases of injury of the head in terms of injury of the brain and degrees of injury of the brain rather than in terms of fracture of the skull.

I agree with Dr. Sachs absolutely in his remarks. In a large series of injuries of the head one encounters patients who present what we would call concussion. We feel, however, that the patient who is rendered temporarily unconscious as the result of a blow, and who on repeated neurologic examinations and observation over days and weeks fails to show any evidence of organic disturbance of the central nervous system, who has no blood in the spinal fluid, and who shows no evidence of rapidly forming edema (such as Dr. Sachs mentioned), one whom we are able to dismiss as absolutely symptom-free, may be a case of concussion.

The fact that those patients who were called "concussion cases" died, is evidence that they had contusions rather than concussions.

DR. FRANKLIN G. EBAUGH, Denver: The behavior disorder syndrome following trauma is analogous to that of acute epidemic encephalitis. Recently I found a similar behavior syndrome following cases of acute chorea. This would fit in well with the pathologic condition that Dr. Osnato has demonstrated this morning.

From the point of view of management, I feel that much can be done by reeducating the parents, particularly by desensitizing them and avoiding a prolonged period of invalidism that is caused by their oversolicitous and oversympathetic attitude toward these children.

DR. OSNATO: Dr. Jelliffe's observation coincides with several that have come within my knowledge. Whether it is merely a coincidence or not, of course it is hard to say, but it is interesting that a number of observers have recorded that soon after injury of the head epidemic encephalitis has developed. I can recall two or three such cases. In some of them, the judgment of those laymen who had the patients in charge for adjustment was sympathetic to the point of view, namely, that the injury lighted up, as they say, the latent process, or set the soil for its later development.

Dr. Davis is quite right. I think that all of us who do Bárány tests regularly in a routine way find great variations in apparently normal persons. Especially at the first examination, anything that tends to disturb the emotional state of a patient will produce hyperirritability. It is only by frequent testing that one can really safely say that there is or is not hyperirritability of the vestibular apparatus. But in many of these patients who regularly showed this hyperexcitability, it is felt that perhaps there was something organic at the basis of the situation.

Of course, when there is a dead vestibule or there is after-turning nystagmus of only four or five seconds, and no response from the vertical canals, accompanied by deafness, the evidence points toward a definite injury to the semicircular canals.

Concerning Dr. Sachs' remarks, the meninges in this case showed only hyperemia and hemorrhage. Dr. Cassasa tells me he did the autopsy. There was some hemorrhage into the meninges, especially marked at the tips of the temporal and frontal lobes, as there nearly always is in these cases, but no gross damage to the brain itself.

A great many people have concussion of the brain and apparently recover completely. This is obvious if one considers the number of young men who

are "knocked out" during football and other games, and in the accidents that occur in industry. The majority apparently recover quite completely, so far as one can determine.

Whether the pathologic change reaches the point seen in our slides in any great number of cases is extremely doubtful. It is perhaps only the exceptional case that shows anything like what was seen in these sections.

However, in most patients who persistently complain and have some objective symptoms, it seems fair to say that there is an organic structural basis. Even the patient having ordinary concussion will often complain of headache and dizziness; and there is frequently a character change also evidenced by extreme irritability and fatigue and a tendency to disturbance of sleep. In these cases, there is no question that there has been some pathologic change similar to what we have shown in this case, with later the laying down of diffuse areas of gliosis and perhaps even cellular changes.

MASSIVE CEREBRAL HEMORRHAGE

ITS RELATION TO PREEXISTING CEREBRAL SOFTENING *

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It is believed that there is a series of events which precipitate the so-called cerebral apoplexy and which seem to hold a secure place in the present conception of spontaneous cerebral hemorrhage. This is well reflected in the descriptions in the textbooks of the factors and events of such vascular accidents. It is commonly held that a miliary aneurysm breaks and that from such ruptured aneurysmal vessels blood rushes suddenly out with a force that is destructive to the surrounding tissue. The tissue is torn apart, pushed aside and rapidly demolished, with the result that a large cavity filled with blood is created. Thus, the so-called hemorrhagic cyst is formed.

This conception of spontaneous cerebral hemorrhage derived its main strength from the monumental contributions of Charcot and Bouchard.[1] The "miliary aneurysms," which they[2] described as the almost constant changes in the vessels found in the brains of those who succumb to spontaneous cerebral hemorrhage, still form the nucleus of every discussion on cerebral apoplexy and are frequently the subject of new investigations.

In recent years, however, many objections have been raised against such an interpretation of the mechanical factors involved in spontaneous cerebral hemorrhage, for it has been felt that many problems, such as those relating to the shape, extent and location of the hemorrhage on the one hand and the changes in the surrounding tissue on the other, have not been satisfactorily solved. Foremost among the recent contributions that are highly significant in their bearing on these problems are those of

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1. Charcot, J. M., in collaboration with Bouchard, M. C.: Hémorrhagie cérébrale, in Oeuvres complètes **9**:3, 1890.

2. Charcot, J. M.: Leçons cliniques sur les maladies des vieillards et les maladies chroniques, Paris, 1867 (troisième leçon, page 48). Bouchard, C.: A Study of Some Points in the Pathology of Cerebral Hemorrhage (English translation), London, MacLachlan and Steward, 1872.

Ellis,[3] Pick,[4] Rosenblatt[5] and Westphal.[6] Not only do these authors fail to corroborate the observations of Charcot and Bouchard, but, surprisingly enough, they come to directly opposite conclusions. In their experience, the so-called miliary aneurysms are not true aneurysms. Their studies lead them to conclude that there is no such selective or, we may say, essential disease of cerebral vessels as was suggested by the French observers. They, like many others, do not deny that aneurysmal dilatations of the blood vessels are frequently found in the hemorrhagic foci, but such aneurysms, in the opinion of the modern and of some of the older pathologists (Koelliker,[7] Virchow[8] and Eppinger[9]), are dissecting aneurysms which are either coincident with or part of the changes that occur in atheromatous vessels. Moreover, it is the belief of the more recent investigators that such aneurysms do not play any particular rôle in the production of cerebral hemorrhage. They regard the hemorrhage as mainly the result of the rupture of diseased blood vessels which may or may not have had aneurysmal dilatations.

Since, therefore, the miliary aneurysms can no longer be regarded as the all important factor in the production of spontaneous cerebral hemorrhage, it is of particular interest now to recall Rouchoux's[10] conception of a "prehemorrhagic" stage of softening which precedes an apoplexy. As early as 1814, he pointed out that in the production of cerebral hemorrhage a previously diminished resistance in the brain tissue near a degenerating blood vessel is an essential and determining factor. A weakened vessel wall and a sudden rise in tension are not enough to produce hemorrhage, but a preexisting area of softening, diminishing the support of the vessels must be present as an additional important and predisposing cause. This idea, though supported later

3. Ellis, A. G.: The Pathogenesis of Spontaneous Cerebral Hemorrhage, Proc. Path. Soc. **12**:197, 1909.

4. Pick, Ludwig: Ueber die sogenannten miliären Aneurysmen der Hirngefässe, Berl. klin. Wchnschr., Feb., 1910, p. 325.

5. Rosenblatt: Ueber die Entstehung der Hirnblutung bei dem Schlaganfall, Deutsche Ztschr. f. Nervenh. **61**:10, 1918.

6. Westphal, Karl, and Baer, Richard: Ueber die Entstehung des Schlaganfalles, Deutsches Arch. f. klin. Med. **151**:1, 1926.

7. Koelliker: Ueber blutkoerperchenhaltige Zellen, Ztschr. f. Wissenschaftliche Zoologie, vol. 1, p. 204.

8. Virchow, R.: Ueber die Erweiterung kleinerer Gefässe, Virchows Arch. f. path. Anat. **3**:427, 1854.

9. Eppinger: Pathogenese der Aneurysmen, Arch. f. klin. Chir. (supplement) **35**:1, 1889.

10. Rouchoux: Recherches sur l'apoplexie, 1814 [cited by Bouchard, footnote 2 (second reference)].

by Todd [11] and a few others, failed for many years to gain general recognition and only recently found support in the contribution of Rosenblatt and Westphal. The last mentioned authors have also realized that cerebral hemorrhage cannot be due solely to two factors (degenerated blood vessels and hypertension) and have raised the question of how to explain the more or less stereotyped and often spherical outline of the hemorrhagic focus in spontaneous bleeding while the experimentally produced hemorrhages tend to irregular, wedge-shaped spread of the injected medium, the latter finding its way along a path of least resistance between fibrous tracts.

If the view that a prehemorrhagic stage exists in the brain tissue before the rupture of a vessel is correct, then it is important to consider the nature of this pathologic process. Rosenblatt, quoting the observations of Baer,[12] points out that in a vast majority of instances of cerebral apoplexy, advanced disease of the kidneys is also present. The latter is either of the atherosclerotic contracted kidney or of the chronic diffuse parenchymatous nephritic type. With this in mind, Rosenblatt postulates the elaboration of a toxic substance by the diseased kidneys, which is in the nature of an enzyme. It is liberated into the blood stream and on reaching selected areas of the brain autolyzes the latter, and produces the prehemorrhagic softening which paves the way for cerebral hemorrhage through the rupture of a diseased and poorly supported blood vessel. It is obvious that such a purely hypothetic explanation cannot be accepted without challenge, and Westphal, realizing the inefficacy of such a theoretical explanation, offers a new idea which also still needs substantiation. He speaks of what he designates as angiospasm, a spasmodic contraction of a cerebral blood vessel, which he believes results in the production of an ischemic zone with the development of an area of diminished consistence in the brain. When the spasm wears off and the vessel opens again to be filled with blood now under higher tension, it meets with a lessened resistance in the brain substance around it, and because of that as well as because of the inherent weakness of the vessel wall it breaks, causing a hemorrhage. This again is a hypothesis which is as yet not adequately supported by well studied material. It is based on the observation of cases of cerebral insult with hemiplegic manifestations, in which the necropsies failed to show anatomic changes which would account for the clinical features. Experimental evidence has also been brought to bear on this hypothesis, but it is as yet too early to accept it as the final word on the prehemorrhagic stage of softening. While this theory cannot be denied and

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11. Todd: Clinical Lectures on Paralysis, p. 126 [cited by Bouchard, footnote 2 (second reference)].

12. Baer, Richard: Apoplexie und Hypertonie, Frankfurt. Ztschr. f. Path. **30**:128, 1924.

while there is sufficient ground justifying further investigation in this direction, we nevertheless feel that in a large number of cases the cause of the primary softening preceding the terminal hemorrhage is to be found elsewhere.

From the study of our own clinical and pathologic material, we have come to believe that a prehemorrhagic stage of softening must antedate the apparently abrupt and explosive vascular insult. The clinical evidence favoring the existence of a prehemorrhagic stage is somewhat circumstantial and is not as convincing as the evidence obtained from the anatomic studies. Still, it seems to us that if clinical observations were made on a larger scale and with greater accuracy, they would yield important confirmatory information. The prehemorrhagic phase of softening frequently escapes clinical recognition because the extent and the location of such a prehemorrhagic area of softening results in so little brain dysfunction that it does not give rise to easily detectable signs, and the symptoms that do exist may not be clearly understood or properly interpreted. In some instances, the gravity of the damage is not fully realized because of the transient character of the symptoms. There is another group of cases that is lost to the investigator because they do not come under the observation of a trained neurologist and are not studied with a view of determining the character of the cerebral involvement but are merely regarded as instances of diffuse cerebral atherosclerosis. If such cases were to be studied with an effort to establish the character and location of the lesion, evidence of a localized destructive lesion might be found.

In this study a limited number of cases are presented in order to illustrate that in nearly every case of cerebral apoplexy there can be found in the patient's history evidence of a preexisting cerebral disease that may have been diffuse or focal in character. In a small number of cases in which no history of previous cerebral disease is obtainable, there is ample pathologic evidence indicating that a diffuse or focal degenerative cerebral process was in existence prior to the terminal event. This pathologic evidence is found in the presence of a more or less highly organized wall around the area of extravasation; in the extent of the destructive process, which seems out of proportion to the short period of time elapsing between the onset of the hemorrhage and the death of the patient, and in the diffuse productive changes of long standing throughout the brain as a whole. Thus we feel that the clinical and pathologic examinations in our series of cases indicate that cerebral apoplexy is a final event in the chain of circumstances, and that it is preceded by an area of softening which existed before the hemorrhage for various lengths of time. This area of softening is probably caused by closure of a blood vessel in the course of a vascular disease of the brain.

REPORT OF CASES

The cases presented here fall into the following groups:

GROUP 1. Cases with precipitate cerebral hemorrhage in which there was no history of previous illness, though there were definite anatomic characteristics indicating widespread, degenerative cerebral vascular disease of long standing.

GROUP 2. Cases in which some clinical evidence of preexisting vascular disease of the brain was present without, however, showing signs and symptoms of focal destructive disease.

GROUP 3. Cases in which distinct histories of preexisting focal disease of long standing were obtained. In this group it is not improbable that the terminal event was provoked by surgical measures.

GROUP 4. A case showing diffuse chronic disease of the cerebral vessels associated with hypertension which, in the absence of preexisting cerebral softening, escaped spontaneous massive hemorrhage.

GROUP 1

CASE 1.—*History.*—E. S., a man, aged 62, was admitted to the hospital, April 6, 1925. He was regarded as being in good health twelve hours prior to admission. He was seen entering his room in the usual way and was apparently well, but a few hours later he was found lying unconscious on the floor and was immediately taken to the hospital.

Examination.—The patient was in coma and cyanotic; the first heart sound was of poor quality. The aortic second sound was accentuated. There were conjugate deviation of the eyes to the right, left corneal anesthesia, left facial paralysis of the central type and paralysis of the right upper and lower extremities with cortical discharges in the extremities on the left side. The abdominal reflexes were absent, with increased deep reflexes on the left side. The Hoffman and Babinski signs were present on the left side, with a suspicious Babinski sign also on the right side. The fundi showed arteriosclerotic changes. The cerebrospinal fluid was blood-tinged and was under moderately increased pressure. The urine showed a trace of albumin. The blood and spinal fluid Wassermann tests were positive. The blood chemical determinations showed urea nitrogen, 27.8; uric acid, 3.5.

Course.—The patient, without regaining consciousness and without any changes in the objective symptoms, died two days after admission.

Necropsy Report.—The pia-arachnoid was dull and presented diffuse areas of discoloration, due to extravasation of blood into the subarachnoid space, and diffuse opacities. The brain was voluminous and showed some asymmetry, the left hemisphere being larger than the right. On palpation, sagging was noted in the left parieto-occipital region. On sectioning, diffuse narrowing of the gray matter and fair dilatation of the lateral ventricles, probably due to general cerebral atrophy, were noticed. Throughout the white substance there were seen many small areas of cystic degeneration. They were particularly large and numerous in the basal ganglia and in the pons. In the left cerebral hemisphere, there was a large excavation extending from about the level of the anterior pillars of the fornix as far back as the level of the parieto-occipital sulcus. It was lined

by a necrotic membrane and contained a small quantity of coagulated blood. It had replaced almost all of the white substance in that region with only a narrow rim of cortex forming its mesial and dorsal boundaries. Free blood was also present in the lateral ventricles. The vessels throughout the brain substance showed advanced atherosclerotic changes, especially at the base of the brain.

Microscopic Anatomy.—Sections from areas of the cerebral cortex showed pronounced gliosis. Fibrous astrocytes formed the predominating type of cells, though other forms of glia cells were also abundant. In the basal ganglia and in various zones of the white substance, small areas of softening were seen. These

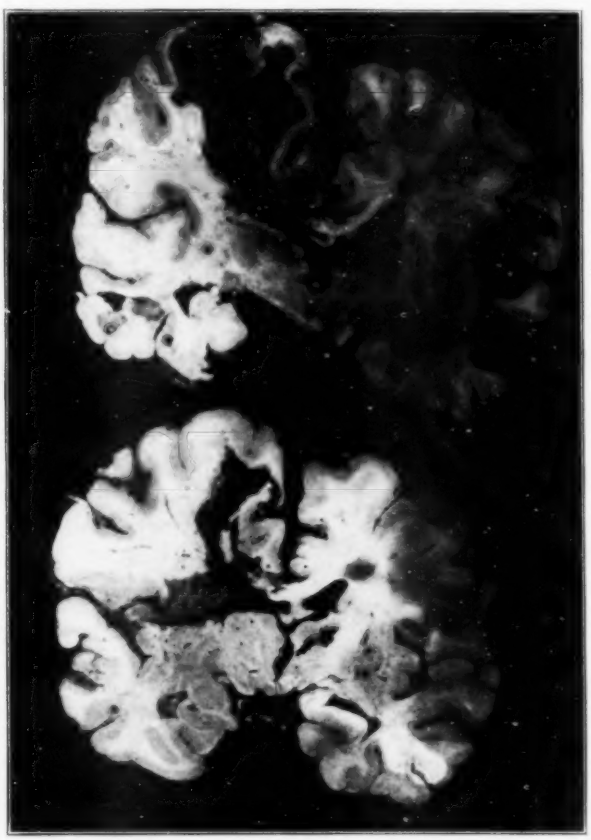

Fig. 1.—Coronal sections of the brain (case 1), showing the hemorrhagic cyst and its free communication with the adjacent portion of the lateral ventricle. Smaller cystic areas of softening are seen in the thalamus opticus and other parts of the white matter.

were in various stages of development. In the more recent areas of softening, the vessels appeared to be prominent because of the loss of brain substance around them. Between these vessels were accumulations of granular cells. About such areas of softening were wide zones of marked gliosis. The wall of the hemorrhagic cyst presented definite features of organization and showed three distinct layers not unlike an abscess wall. The innermost layer consisted of brokendown

brain tissue mixed with extravasated material; the middle layer, vascular in character, contained many newly formed blood vessels surrounded by glial and lymphocytic elements and, finally, the outmost layer was represented by a fairly wide zone of gliosis.

Comment.—The precipitate onset of the illness in a man who was thought to be well is of significance in this case. In spite of the suddenness of the onset and the absence of any evidence in the anamnesis suggesting previous cerebral involvement, the pathologic examination seemed to indicate that the cerebral lesion had had a much longer existence than the clinical history would suggest. The positive serologic symptoms simply indicate the possibility of a syphilitic etiology, but the histologic studies of the brain did not reveal any evidence of active cerebrospinal syphilis.

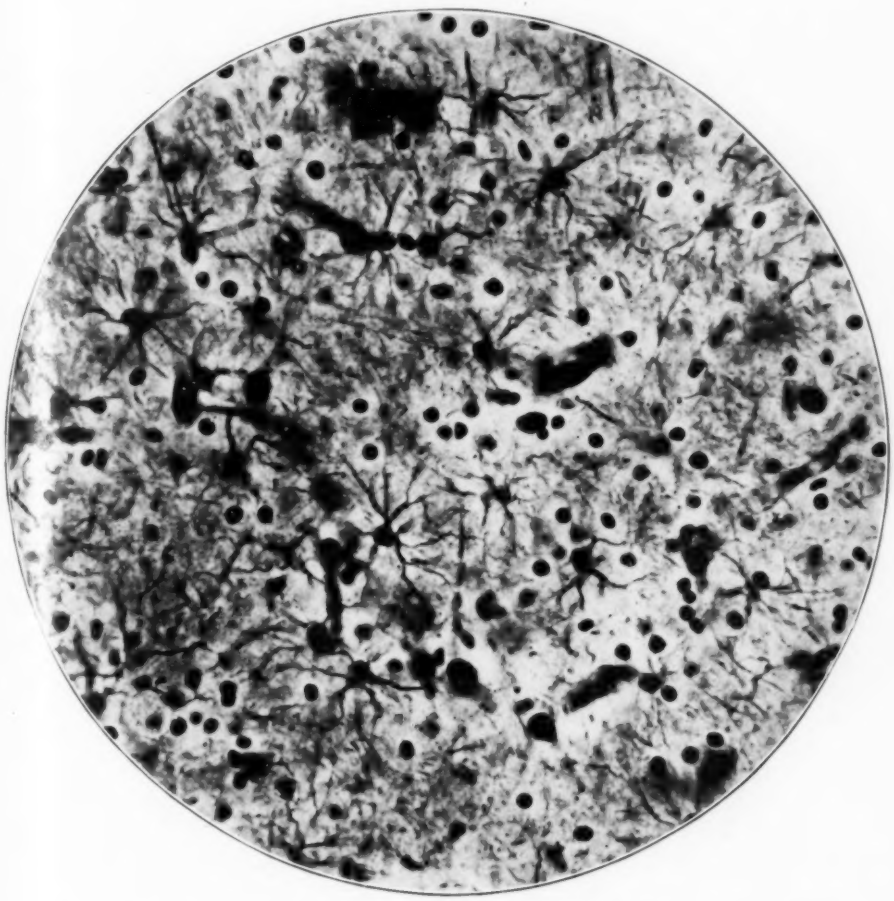

Fig. 2.—Diffuse gliosis in the cerebral cortex in case 1. The fibroblastic astrocytes are the predominating type of glia cells. Hortega's silver carbonate stain (Globus modification); × 350.

Case 2.—*History.*—M. A., a man, aged 68, was admitted to the hospital, Oct. 7, 1925. He was said always to have been well except for the occurrence of urinary frequency, for the relief of which a prostatectomy was performed four years prior to his admission to the hospital. On the day of the onset of the terminal illness, the patient was seen to enter his place of employment apparently in good health, but was not seen again for about three hours. He was then discovered on the floor of the washroom in a state of collapse. He was immediately brought to

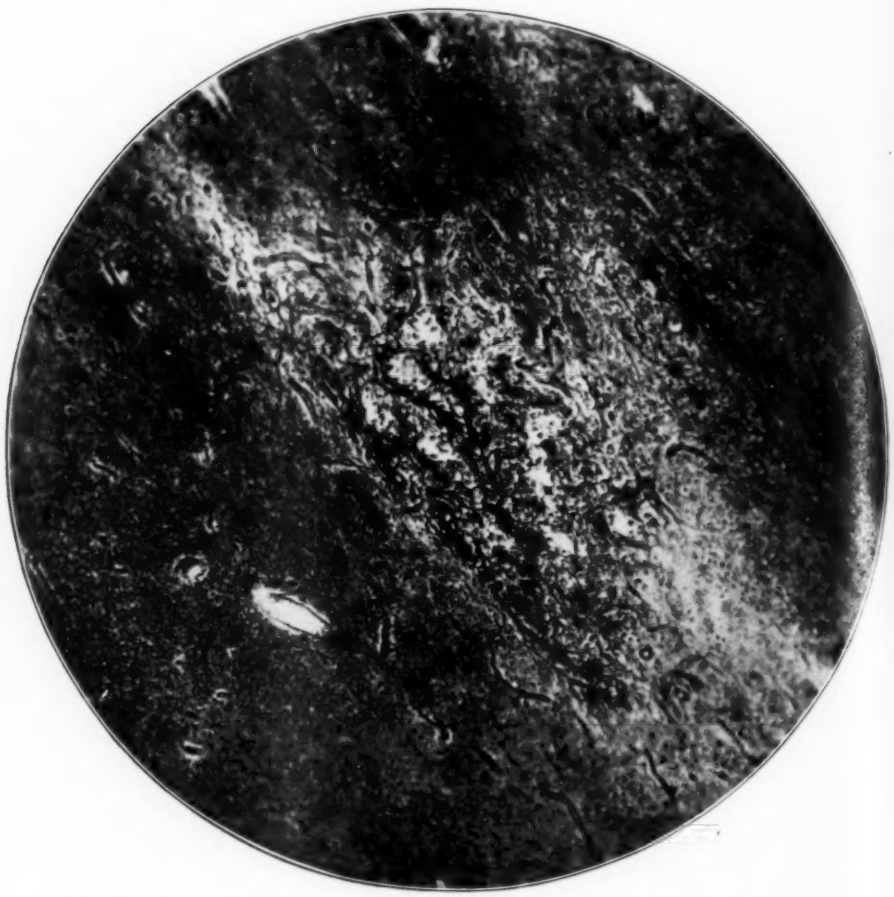

Fig. 3.—A small area of softening, of recent origin, with its blood vessels exposed as the result of the softening. About these blood vessels there are aggregations of granular cells. The area of softening is surrounded by a zone of proliferative gliosis. Hortega's silver carbonate method (Globus modification); ×75.

the hospital, and there he was found to be in deep stupor. He was markedly cyanotic; the pupils were dilated and fixed and the neck was rigid. Generalized spasticity, bilateral Babinski sign, marked hypertension (systolic 230, diastolic 90) and subnormal temperature were present. He died a few hours after admission to the hospital.

Necropsy Report.—In the right temperoparietal lobe, a large subcortical hemorrhage was present. The walls of the hemorrhagic cavity were ragged and showed a defect in the temporal region, permitting the escape of blood into the subarachnoid space. The basal ganglia and the thalamus, on the side involved, showed disseminated areas of softening. The ventricles were filled with blood. On washing out the blood from the hemorrhagic cavity, a fully exposed blood vessel was seen crossing a cavity with many small ruptured branches given off

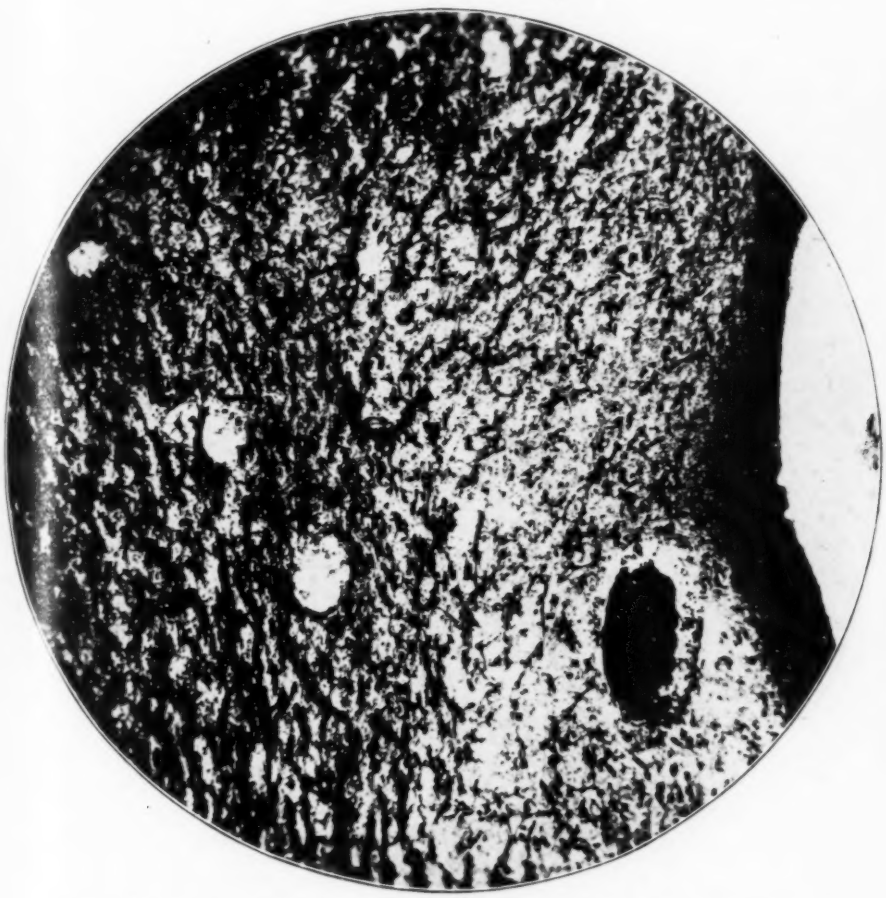

Fig. 4.—A section in the wall of the hemorrhagic cyst with its three distinct zones. There are: an internal zone consisting of a homogenous structureless layer; a middle zone of loose reticular structure in which there are many granular cells, astrocytes and a variable number of blood vessels, and an outer zone of intense gliosis. Hortega's silver carbonate method (Globus modification); × 100.

at its lateral end. The vessels at the base of the brain showed marked atherosclerotic changes, and many small aneurysms were noted in the posterior inferior cerebral artery. The microscopic characteristics in this case are essentially the same as in case 1.

Comment.—Here also we have abrupt onset in a fatal cerebral lesion in a patient who was believed to be well. The gross anatomic examination in this case showed a vast amount of destruction which could not have occurred during the exceedingly short period of the fatal illness. There was ample evidence, in the gross as well as in the microscopic appearance of the basal ganglia, in favor of an old standing degenerative process, and it can be assumed also in this instance that an area of softening and cavitation about a degenerated blood vessel preceded the terminal hemorrhage.

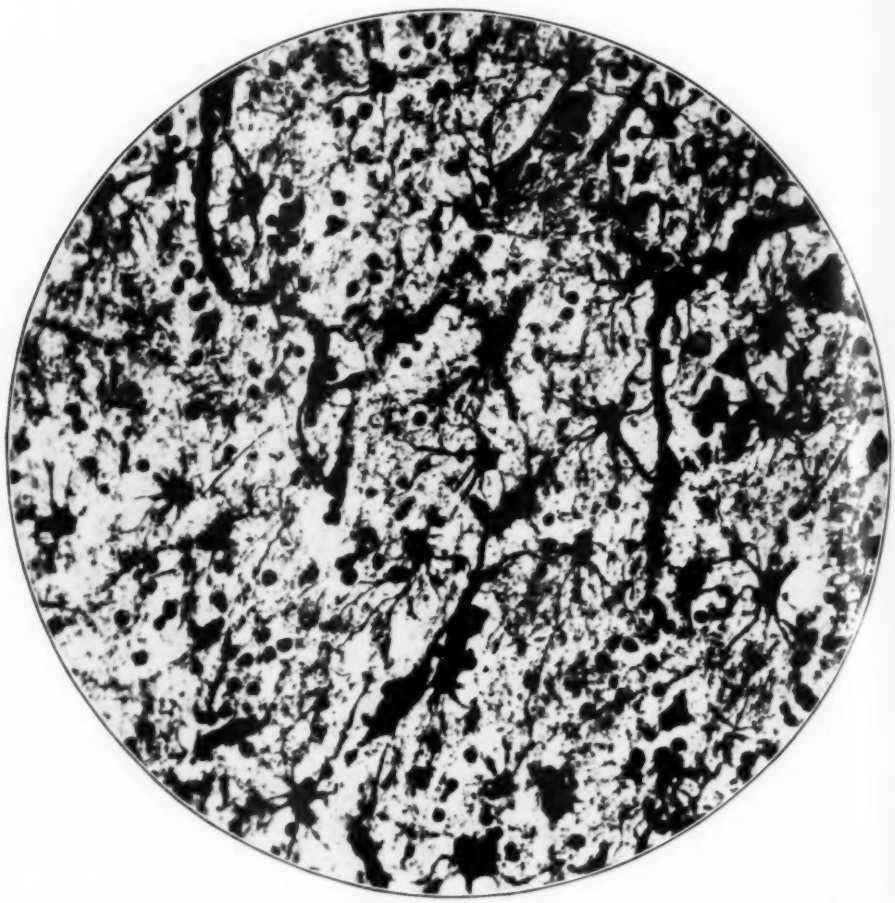

Fig. 5.—The type of glia (fibroblastic astrocytes) in the outer dense zone of gliosis. Hortega's silver carbonate method (Globus modification); × 350.

Here again it may be said that if this patient had been carefully examined at some time before the fatal event, subjective and objective evidence would, without question, have been found of the diffuse destructive process in the regions involved.

GROUP 2

CASE 3.—*History.*—N. B., a woman, aged 58, was said to have been well until ten years preceding the onset of the terminal illness. However, during these ten

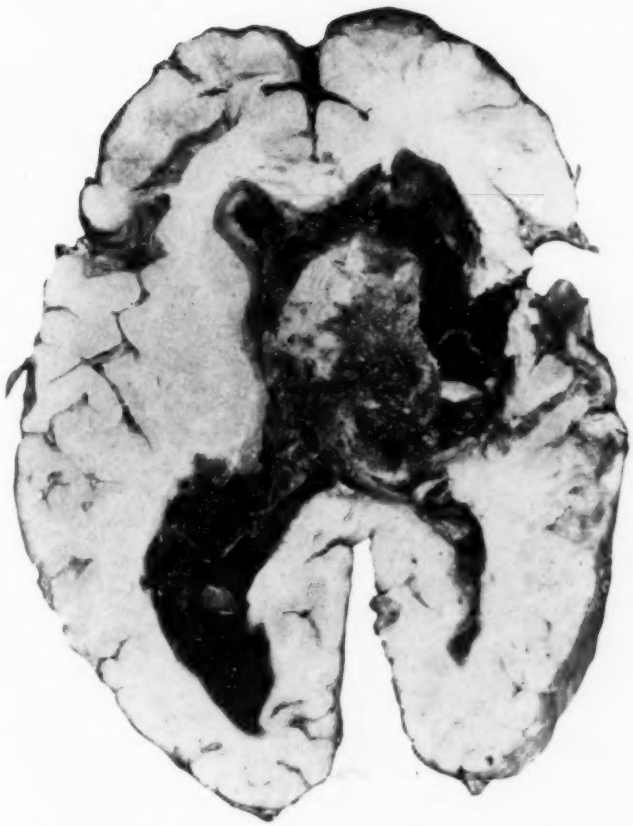

Fig. 6.—Appearance of brain in case 2, showing the marked and diffuse destruction of brain tissue, the naked blood vessel bridging over the hemorrhagic cyst and the free communication of the cyst with the ventricular cavity.

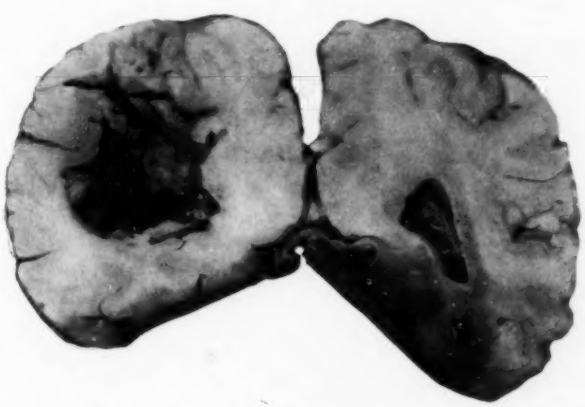

Fig. 7.—Coronal section of the brain in case 3, showing the hemorrhagic cyst. The compressed posterior horn on the same side should be noted.

years she would complain frequently of severe headache, impaired vision, gradual loss of weight, dyspnea on exertion, swelling of the legs and nocturia. Two days prior to admission to the hospital, the patient was seen returning home from shopping in apparently good health, but was soon heard to cry out, and was found lying across the bed unconscious. She vomited and passed into deep stupor.

Examination.—On admission, the patient was in stupor. Râles were heard at both bases. The aortic second sound was accentuated and the radial arteries were

Fig. 8.—Aneurysmal dilatation of vessel in wall of the hemorrhagic cyst. Hortega's silver carbonate method (Globus modification); × 275.

rigid. There was a right hemiparesis with signs of involvement of the pyramidal tract on that side. The abdominal reflexes were not elicited. The blood pressure was 195 systolic and 100 diastolic. The eyegrounds showed arteriosclerotic changes. There was a trace of albumin and granular casts in the urine. The blood Wasserman test was negative, and the blood chemistry showed: urea nitrogen, 11.2, and noncoagulated nitrogen, 38.5. Her condition declined gradually; the pupils became unequal, the left larger than the right, and she died without regaining consciousness seven days after admission to the hospital.

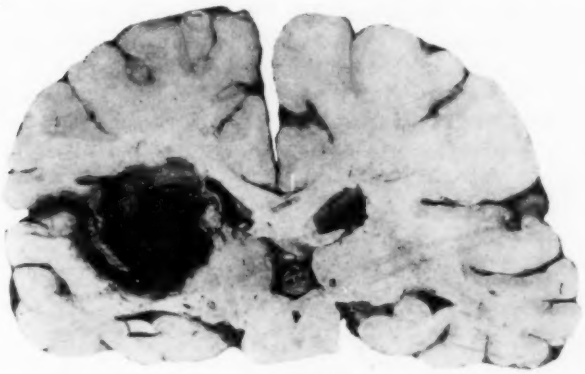

Fig. 9.—Coronal section of the brain in case 4, showing the hemorrhagic cyst with a naked, exposed blood vessel bridging over the cavity. There is slight compression of the ventricle on the ipsolateral side.

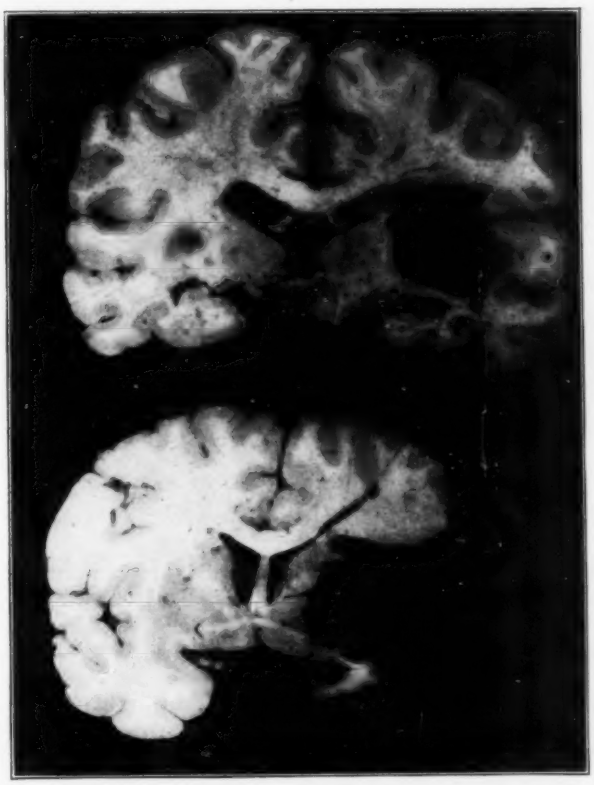

Fig. 10.—Coronal section of the brain in case 5, showing the hemorrhagic cyst and the many small, degenerative, cyst-like areas in the thalamic region.

Necropsy Report.—A small quantity of fluid blood was found in a thin layer underneath the dura over the left hemisphere, in the region of the parieto-occipital lobe. The blood was easily removed, leaving here and there small clots adherent to the dura, or to the underlying pia-arachnoid. A small amount of blood was also found in the subarachnoid space. This extravasated blood apparently escaped through two small erosions of the cerebral cortex in the posterior portion of the occipital lobe. Pressure in that region caused further escape of blood from the

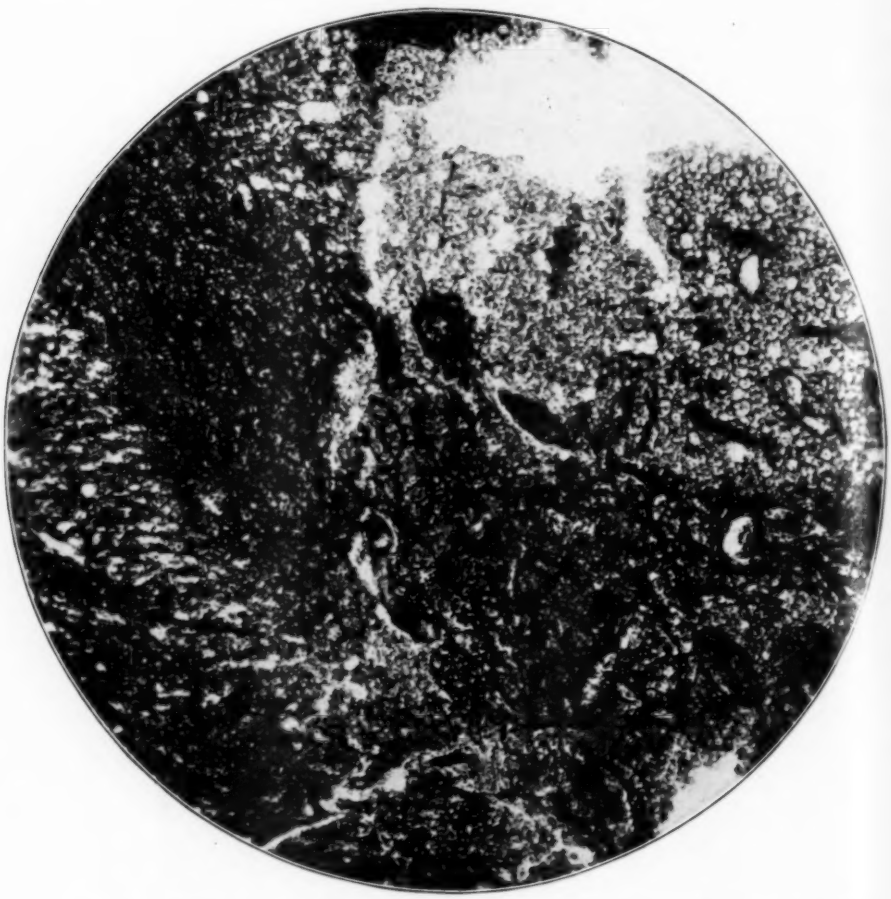

Fig. 11.—Earlier stage in the organization of the hemorrhagic cyst wall. Here also three distinct zones are seen: (*A*) some of softening; (*B*) vascular zone; (*C*) zone of gliosis. Hortega's silver carbonate stain (Globus modification); × 70.

interior of the hemisphere. On incision of the left hemisphere, a large cavity filled with blood was found It extended from about 1 inch (2.5 cm.) anterior to the occipital pole forward as far as the level of the postcentral sulcus. In width it involved almost all of the white substance of the hemisphere and encroached posteriorly on the posterior horn of the lateral ventricle. The cavity was lined by a well defined zone of grayish-brown necrotic tissue, with numerous apparently

obliterated vessels projecting into the cavity. A short distance from the wall of the hemorrhagic cavity was found a small cyst filled with a colorless gelatinous substance. The hemorrhagic cyst was separated from the lateral ventricle by a thin septum of soft tissue. The microscopic examination in this case was not unlike those described in the two previous cases. In this instance, however, a small aneurysmal dilatation of a blood vessel was found exposed in the cystic wall.

Fig. 12.—A thrombosed blood vessel in the proximity of a hemorrhagic cyst. The vessel is surrounded by dense glial fibrosis. Hortega's silver carbonate stain (Globus modification).

Comment.—This case differs somewhat from the preceding ones in that there was a long history of headache, impaired vision and other manifestations pointing to the existence of cardiovascular disease, with probable cerebral involvement, although at no time were signs of a focal cerebral lesion observed. It is, however, likely that if the patient had been examined by a neurologist at some time before the fatal illness, evidence of a focal destructive lesion would have been found.

Case 4.—*History.*—S. N., a woman, aged 45, was admitted to the hospital, Feb. 25, 1925. She was apparently in good health up to six weeks prior to admission, except that she was known to have had high blood pressure for a period of seven years preceding the illness. Six weeks previously, while at work, she suddenly became dizzy and fell to the floor. She lost consciousness for a short time, vomited repeatedly and on examination by a physician showed a complete left sided paralysis. For the next four weeks, she showed slight improvement, but at the end of that time she suddenly became delirious, passed into stupor and lost the little gain she had made.

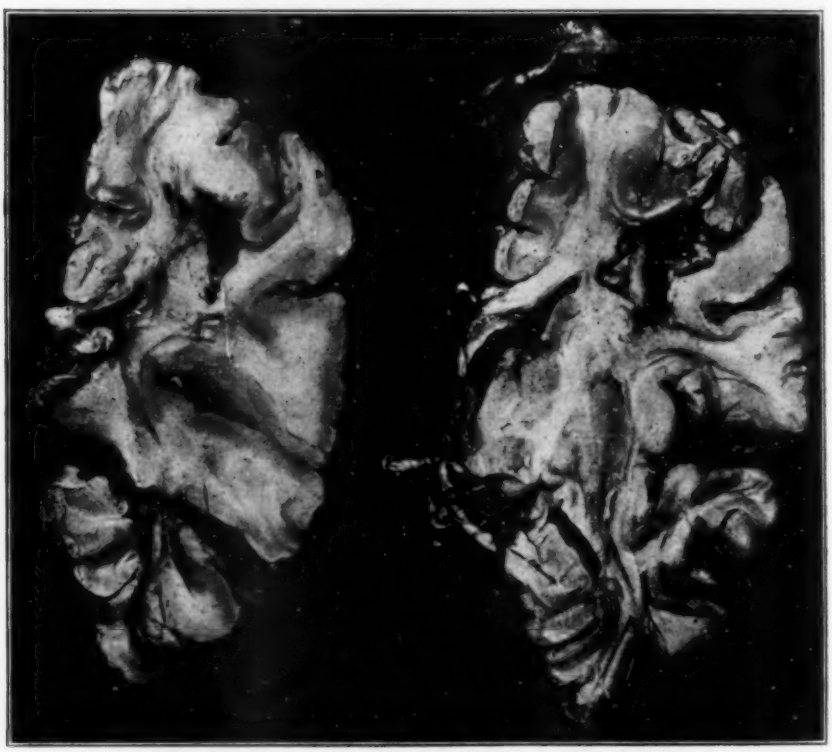

Fig. 13.—Section of brain, showing location and extent of the hemorrhagic cyst.

Examination.—The patient was poorly nourished. The pupils were equal and reacted well to light and in accommodation. There was conjugate deviation of the eyes to the right, slight blurring of the nasal margins of the disks and a left hemiplegia. The cerebrospinal fluid was clear, colorless, under increased pressure and contained 39 cells per cubic millimeter.

Course.—The patient showed mild oscillations in her mental condition; periods of stupor alternated with lucid intervals, and because of these the question of a cerebral neoplasm was raised. A ventriculography was performed but with negative results. The patient died eighteen days after admission to the hospital.

Necropsy Report.—In the depth of the sylvian fissure of the right cerebral hemisphere was a large fluctuating mass covered by a thin layer of cerebral cortex.

On sectioning the brain, a large cavity was found, replacing almost all of the island of Reil and a major part of the adjacent basal ganglia. The cavity was filled with coagulated blood and was separated by only a narrow zone of necrotic tissue from the body of the lateral ventricle. The wall consisted of a wide zone of necrotic tissue. A medium sized, naked blood vessel crossed the cavity and gave

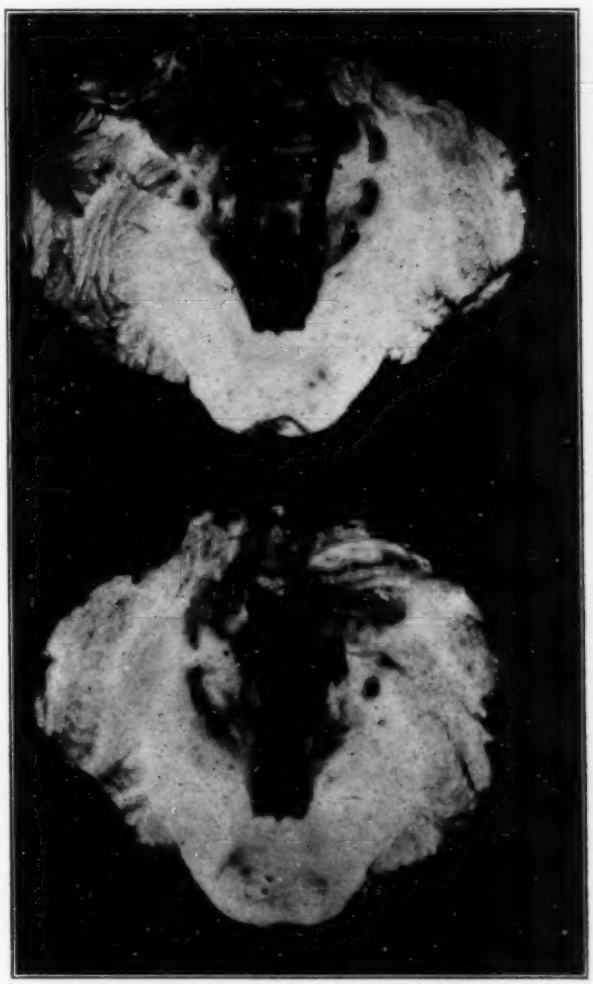

Fig. 14.—Section of cerebellum, showing location and extent of hemorrhagic cyst and other areas of softening.

rise to several small branches which were ruptured. The optic thalamus on the same side showed many areas of degeneration. Similar areas of degeneration were also seen in the pons.

The microscopic observations were similar to those already described in the other cases, although the generalized gliosis was less marked throughout the cerebral cortex.

Comment.—This case differs from the preceding three cases in that the patient was known to have had hypertension for a period of seven years preceding the terminal illness and in that the fatal illness lasted about nine weeks. This history, taken in conjunction with the anatomic symptoms, indicates that the cerebral disease was slowly progressive in its development.

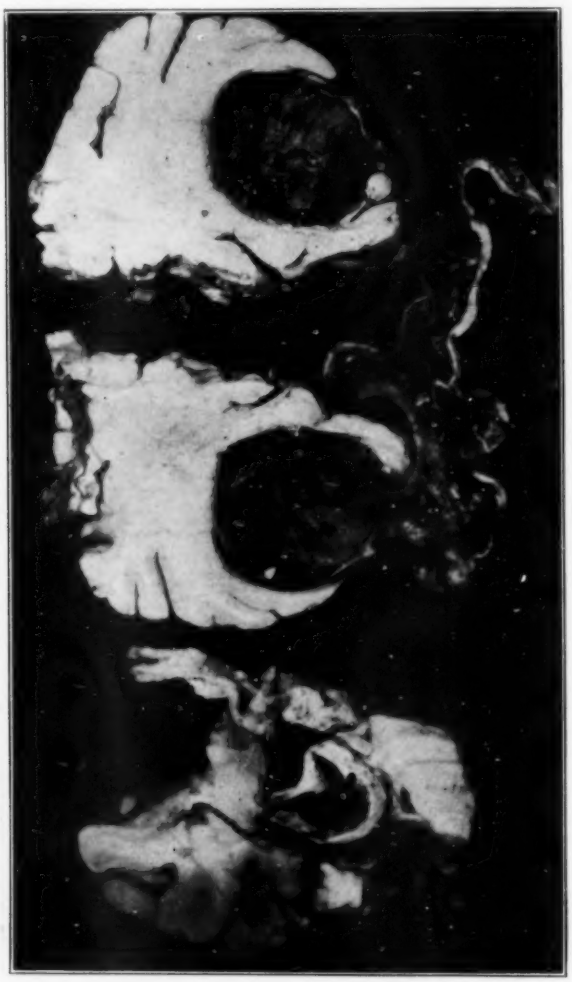

Fig. 15.—Section of brain, showing the aneurysmal cyst embedded in the brain substance without apparent cerebral softening.

It seems obvious that the focal lesion existed at least for a length of time corresponding to the duration of her fatal illness and it is not too speculative to assume that this cavity, resulting from the softening, prepared the way for the terminal hemorrhage.

Case 5.—*History.*—A. N., a woman, aged 42, was admitted to the hospital, March 24, 1925. For about eight years preceding the onset of the terminal

illness, she had complained of severe headache and during the last three years she was found to have a high blood pressure. During the latter period she complained of impairment of vision. Nine days prior to admission to the hospital, she suddenly collapsed and fell to the floor. Her husband, who soon came to her aid, noted complete loss of power on the left side of her body. She did not lose consciousness and complained of pain in the paralyzed side. She was immediately taken to the hospital, where she soon lapsed into a state of semistupor which was occasionally interrupted by semilucid periods.

Examination.—The patient was dull, apathetic, did not cooperate, and could not be made to answer questions. Her pupils were unequal, the right being larger than the left, but both reacted to light. Corneal anesthesia, left external rectus weakness, left hemiplegia, involving the extremities and the face, and slight rigidity of the neck were present. There was also a left hemianalgesia. The right optic disk showed blurred margins, and there were frank atherosclerotic changes in the retina of the left eye. The cerebrospinal fluid was clear and yellowish, under normal pressure and contained 5 cells per cubic millimeter. Blood pressure was 190 systolic, 140 diastolic. The heart was enlarged.

Course.—The age of the patient was somewhat against the diagnosis of a vascular lesion of the brain, and in order to exclude an intracranial tumor a ventriculography was performed. The results were unsatisfactory, but the cerebrospinal fluid removed in the course of the ventricular puncture was found to be bloody. Following the ventricular puncture, the patient passed into deep stupor and died on the fifteenth day of her stay in the hospital.

Necropsy Report.—The subarachnoid space showed an increased amount of clear, amber-colored fluid. The cerebral convolutions were flattened, particularly in the right frontoparietal region. On sectioning of the brain, a cavity was found occupying the posterior two thirds of the right frontal lobe and the adjacent third of the parietal lobe of the right side. The cavity was filled with coagulated blood. It was lined by a rough and necrotic membrane. The vessels at the base of the brain were markedly thickened and tortuous, and showed many atheromatous plaques, particularly throughout the course of the right middle cerebral artery.

On removal of the blood from the cavity by repeated washing, it was found that in its anterior portion, the hemorrhagic cyst was subdivided into two separate compartments, each lined by its own membrane. Within the cyst naked blood vessels could be seen. The cyst was separated from the adjacent portion of the lateral ventricle by an exceedingly thin wall.

Microscopic Anatomy.—In addition to the histologic changes common to the cases already described, this case presented an earlier stage in the organization of the hemorrhagic cyst wall (fig. 4). Here also three distinct layers were discernible: (*a*) a layer of softened, disorganized tissue intermingled with numerous gitter cells; (*b*) a vascular layer in which astrocytes, granular cells and mononuclear elements participated to an almost equal degree, and (*c*) finally a zone of gliosis. In an adjacent area of the brain a large thrombosed central blood vessel was found. It can be assumed with a fair degree of certainty that it was this or other similarly obliterated vessels that were responsible for the area of softening—and the ultimate hemorrhage.

Comment.—In this case, as in case 4, there is clinical evidence of antecedent cerebral disease followed by a somewhat protracted fatal illness. Here also it can be assumed from the anatomic observations that a latent cerebrovascular disease, with focal encephalomalacia, prepared the ground for the terminal event.

GROUP 3

CASE 6.—*History.*—U. S., a tailor, aged 45, was admitted to the hospital, April 18, 1923. The patient had been taken ill four years before, when he had suddenly developed paralysis of the right side of the face. Power returned at the end of four days, but following this attack a change in personality was noticed. He became restless, excitable and at times irrational. His vision began to fail and he had to give up work. Somewhat later, occasional incontinence of urine developed. It was noted that he dragged his right leg. A tremor developed in the right arm. He became emotionally unstable; his memory began to fail and his general condition was rapidly declining.

Physical Examination.—The patient was apathetic, and it was difficult to hold his attention. The pupils were equal, and reacted well to light and in accommodation. The left fundus showed marked engorgement of the veins, but they were not tortuous. A slight facial weakness, slight weakness of the right leg and ataxia and adiadokokinesis of the left arm were present. All deep reflexes on the right side were more active than those on the left. The Babinski sign was present on the right side. The right abdominal reflexes were diminished. The blood pressure was 170 systolic, 105 diastolic. The cerebrospinal fluid was clear and under normal pressure and contained 40 cells per cubic millimeter. The blood and spinal fluid Wassermann tests were negative.

Course.—The diagnosis was uncertain. It was difficult to decide between a vascular lesion and a neoplasm in a person with a definite hypertension. Ventriculography was performed and showed no displacement or change in the form of the ventricles. The significance of this was not appreciated at that time; the possibility of a neoplasm was still considered, and an exploratory craniotomy was recommended. This was done in the left frontal region. An aspirating needle passed into the left frontal lobe brought a moderate amount of blood, suggesting a deep lesion situated somewhat posterior to the motor area. No tumor was found. After the operation, the condition of the patient declined rapidly, and death occurred on May 13.

Gross Anatomy.—The left cerebral hemisphere appeared somewhat collapsed and flattened, but showed no evidence of increased intracranial pressure. On incision of the left hemisphere, a large cavity was found situated beneath the precentral and postcentral convolutions. The cavity was collapsed and contained a small quantity of blood. It had a rough, irregular lining. There were also other areas of softening, yellowish in color and apparently old, in the posterior end of the left lenticular nucleus. The vessels at the base of the brain, including the entire circle of Wills, showed advanced atherosclerotic change.

The microscopic anatomy did not differ materially from that described in the preceding cases.

Comment.—In this case there is a history of a focal as well as a generalized cerebral lesion of four years' duration, with a prolonged remission in the focal manifestation but with gradual accentuation of the symptoms pointing to a generalized process. The assumption is justified in this instance that the focal lesion which was found at postmortem is identical in location and probably in character with that which precipitated the first hemiplegic insult.

It will be recalled that the patient's entrance into the hospital was not provoked by any acute change in his status which could be attributed to hemorrhage, but because of his gradual decline, mainly in the intellectual sphere. The surgical steps were undertaken in the belief that the gradual course might indicate an atypical cerebral neoplasm. It is most probable that here again the fatal cerebral hemorrhage was a terminal event, occurring as the result of a trauma in the

course of the passage of a needle into a preexisting softened area. Ordinarily, the passage of a needle through the brain substance does not cause massive hemorrhage. It may be assumed that if there had not been an area of antecedent softening, the hemorrhage would not have ensued.

Case 7.—*History.*—R. R., a woman, aged 25, was admitted to the hospital, July 7, 1924. She had been under observation in the hospital on three different occasions. On her first admission, April 14, 1920, she gave a history of having had an attack of acute nephritis three years previously. She was said to have made a good recovery, but she soon began to complain of frequent dull headaches, and evidence of hypertension developed. The symptoms grew progressively worse, and shortly before the final admission to the hospital her condition became markedly aggravated by greater intensity of headache, frequent attacks of vertigo, dyspnea and dull pain in the precordial region.

First Admission.—Physical examination at this time revealed definite enlargement of the heart. The heart sounds were loud and booming with a short systolic murmer at the apex, and a ringing second aortic sound. The urine showed a trace of albumin. The blood Wassermann test was negative. The blood chemical determinations were within normal limits. The blood pressure was 195 systolic and 125 diastolic. The fundi showed definite arteriosclerotic changes. The patient was thought to have chronic interstitial nephritis with hypertension. She remained in the hospital for a short time, and went home in an unimproved condition. Her condition remained stationary for about four years, until six weeks before the second admission to the hospital on June 5, 1924, when a sudden change took place. Her headache became severe and almost constant, and she began to vomit frequently. Her vision became impaired and continued to fail and weakness in the extremities developed.

Second Admission.—At this time, the patient presented the following symptoms: The head was held with a left cerebellar tilt; there was a slight tremor of the head, slight ptosis of the left upper eyelid, bulging of the left eye, slight horizontal nystagmus to the right and bilateral corneal hypesthesia. The pupils were irregular and unequal; the left pupil reacted poorly to light and the right was not prompt; both pupils reacted well in accommodation. The jaw was drawn to the left and downward, and there was a left facial weakness. There was left adiadokokinesis and cerebellar form of the hands, which was more marked on the left side. There was ataxia in the left arm, faulty recoil in both upper extremities and weakness in both arms. The reflexes in the upper and lower extremities were greater on the right than on the left side. Hearing was impaired on the left side. Bilateral papilledema was present.

The symptoms and signs suggested the diagnosis of a neoplasm in the left cerebellar hemisphere, and suboccipital decompression and exploration of the posterior fossa was performed. No neoplasm was found, but marked arteriosclerotic changes in the vessels on the surface of the cerebellum and a peculiar yellowish discoloration of the left cerebellar lobe were noted. There was no change in consistence, and no fluctuation in that lobe was felt. Following the decompression, rapid improvement took place, as shown by the subsidence of the objective neurologic symptoms, disappearance of the headache, marked reduction in the swelling of the optic disks, return of power in the extremities and return of normal station and gait. The patient remained fairly comfortable for about two weeks, after which headache, dizziness, vomiting and emotional instability reappeared, with periods of irrationality.

Third Admission.—The patient was readmitted to the hospital, July 7, 1924. She had developed motor aphasia, staggering gait, tendency to fall to the left,

bilateral external rectus weakness; left corneal hypesthesia; left facial weakness; deviation of the tongue to the right; hyperreflexia on the right side; diminution of right abdominal reflexes; bilateral Babinski sign; adiadokokinesis on the left side, and bilateral papilledema going on toward atrophy. The blood pressure was 250 systolic and 170 diastolic. In view of the aphasia and previous negative symptoms on suboccipital exploration, the possibility of a left frontal neoplasm was considered and ventriculography was undertaken for the purpose of localization. The patient began to fail soon after the injection of air and died a few hours after the operation. The necropsy was limited to the cranium.

Necropsy Report.—The pial vessels presented numerous atheromatous areas in the form of narrow, yellowish rings, somewhat raised on the surface of the vessels. The brain showed marked flattening of the gyri. When the brain was removed, the cerebellum appeared firmly attached to the overlying meninges, making it necessary to break the adhesions forcibly. In this way, parts of the posterior lobe of the cerebellum which were attached to the meninges were torn, opening a large cavity filled with blood and extending deep into both cerebellar hemispheres. The left cerebellar hemisphere, however, seemed to be more involved than the right. The vessels at the base of the brain and their tributaries and branches showed marked atheromatous changes. The vertebral arteries, however, were most markedly thickened, the right being almost occluded. Not a single vessel was found free from changes.

On sectioning of the brain, the following alterations were found in the cerebellum: a section through the middle cerebellar peduncles presented a large cystic cavity, elliptic in outline, with the long axis in the dorsoventral directions. It was about 4 cm. long by 25 cm. wide. Its lining was rough, granular and dark brown in appearance, and was not well demarcated from the adjacent brain tissue. Hard coagulated blood was found adherent to certain areas of the wall of the cyst. There was softening near the cyst, with small excavations here and there about its wall for a distance of about 1 cm. Small hemorrhagic foci were also seen in the vicinity of the cyst. These hemorrhagic foci and the areas of softening gave a moth-eaten appearance to structures adjacent to the cyst. There was direct communication between the cyst and the fourth ventricle. The latter was almost completely filled with a dark brown, hard thrombus of fairly recent origin. The aqueduct of Sylvius was also completely occluded by a similar mass of hardened blood, while the third and lateral ventricles, which were definitely enlarged, were filled with liquid blood containing small, recent thrombi.

Other areas of softening of various sizes were found, most extensively in the left putamen. The lenticular nuclei were extremely soft, in marked contrast to adjacent portions of the brain.

Comment.—There can be little doubt in this case that the hemorrhage was a terminal event. There was also evidence that softening in that area preceded the fatal hemorrhage. The symptoms of increased intracranial tension and the improvement following the first decompression are unusual in the clinical course of a degenerative lesion. As in this case the lesion was situated around the fourth ventricle, it is fair to assume that edema had caused an obstructive hydrocephalus with its attendant symptoms.

GROUP 4

CASE 8.—*History.*—I. S., a woman, aged 59, was admitted to the hospital, June 12, 1924. She had had seven children, most of whom had had frequent nose bleed. Thirty years before admission, at the age of 27, for a period of two years the patient had a few infrequent so-called epileptic attacks. At the end of this period she remained free from such attacks until two years before admission,

when there was a return of the seizures. These had since occurred at the rate of one attack every three or four months. For the past twenty-five years, the patient also suffered with kidney trouble, which developed in the course of pregnancy. At that time she had an acute illness of five or six weeks, passed bloody urine, had swollen feet and a period of unconsciousness. She also had had frequent nose bleed. The present illness dated from two months before admission, when the convulsive attacks became frequent, occurring from one to four times weekly. The seizures were sudden in onset; the patient would fall to the ground; the body would become rigid and would shake. The eyes were glassy, the face cyanotic and froth would appear at the mouth. The attacks lasted for about twenty minutes; afterwards, the patient appeared dazed and complained of severe pain in the back of the head. For twenty-four hours before admission the attacks had become still more frequent, occurring almost every half hour. Between attacks the patient was in a stupor and could be aroused only with difficulty.

Physical Examination.—The patient was in coma. There was no uremic odor. The pupils were unequal; the left was dilated and fixed, and the right was contracted and reacted sluggishly. Jacksonian convulsive attacks occurred every half hour. The twitchings began on the left side of the face and spread to the arm and leg of the same side and then to the right side. The contractions were clonic in character, and were more marked on the left than on the right side. There was apparently no loss of motor power; all deep reflexes were active, on the left more than on the right; the abdominal reflexes were absent; the Babinski sign was present on both sides. The left optic disk had a clear margin and marked pallor; the vessels were thin walled and not tortuous. The blood pressure was 140 systolic and 77 diastolic. The laboratory examination showed: blood Wassermann reaction, negative; blood chemistry: urea nitrogen, 23.1; noncoagulable nitrogen, 51; uric acid, 31; creatinine, 1.1. The cerebrospinal fluid was clear, under increased pressure, and contained a few red blood cells.

Clinical Course.—For the first few days in the hospital, some improvement was noted. The patient became more alert and cooperated better. A definite left hemiparesis developed, and the left pupil remained large and fixed. Convulsions occurred less frequently but were of longer duration. They were more marked on the left side.

Epilepsy, encephalitis and multiple neoplasms, were considered in diagnosis.

On June 14, it was noticed that the patient's condition was becoming worse. The convulsive seizures had again become more frequent. They were preceded by vertigo and began in the left side of the face, the mouth being drawn to the left, the eyes turned to the left, the left eyelids closed and the left arm and leg slowly flexed. Consciousness was not completely lost; the patient could remember questions asked during the attack. Directly after an attack, the left hemiparesis was more marked. The frequency of such attacks, their jacksonian character and the fairly good localizing signs pointed to a right frontal lesion. A roentgen-ray examination showed marked dilatation of the right anterior diploic veins merging into a calcareous mass lying in front of the motor area.

On June 20, an exploratory craniotomy was done, and several tumor masses were found on the surface of the cortex, each globular in appearance and enveloped by a thick capsule; another small mass could be felt in the depth of the brain substance. The patient died, June 21, twenty-four hours after the operation.

The necropsy was limited to the cranium.

Necropsy Report.—The right frontal lobe was definitely smaller than the corresponding portion of the other hemisphere; the gyri were atrophic and were

thrown into supernumerary folds. On the surface near the dorsal border was a large vessel growing forward and parallel to the dorsal border and terminating in a small sac about 1 cm. in diameter. The sac was embedded in a depression about 5 cm. posterior to the frontal pole. When the sac was elevated, a depression was found, lined by smooth and glistening pia. Directly posterior to the sac there was another elevation, about 1.5 cm. in diameter on the free surface. On cutting into the brain at this point, another larger, encapsulated mass was revealed. This was oval in outline; it was 3 cm. in the long diameter, and was embedded deep in the substance of the frontal lobe. A cut section of this mass showed it to be hard, somewhat brittle and grayish-brown; it gave the impression of an organized thrombus in an aneurysmal cyst. Directly posterior to this, extending back as far as the postcentral gyrus, there were several similar cysts varying in size from 0.5 to 2 cm. in diameter. The smaller cysts were also well encapsulated and were surrounded by thick, fibrous, calcified walls. Their cavities were filled with yellowish, granular material and a small amount of yellowish fluid. The inner lining of the walls, however, was smooth, grayish-white and glistening. All the cysts were easily dislodged from the substance of the brain, leaving behind smooth surfaces of only moderately softened brain substance. All these cystic structures were aneurysmal dilatations of calcified vessels, though only one, the most anterior cyst, could be traced to a vessel, the wall of which appeared to be continuous with that of the cyst. In the others it is probable that they arose from vessels, but the evidence of such a relationship had been lost by a chronic obliterative process.

Comment.—This case illustrates that hypertension and diseased cerebral vessels are in themselves insufficient to produce cerebral apoplexy if the consistence of the brain remains unmodified.

From the history of the case it may be assumed that the aneurysms of the vessels are of old standing. They have likely appeared at an age when the patient's vessels were more elastic and when there was better collateral circulation in the brain. Hence, softening did not occur; instead, the brain substance adjusted itself to the aneurysm as to foreign bodies without softening, hence wihout subsequent hemorrhage.

SUMMARY AND CONCLUSIONS

Anatomic and clinical observations are here assembled in support of the belief that spontaneous massive cerebral hemorrhage is a terminal phase in a sequence of events which have their beginning in a generalized or somewhat localized disease of the cerebral vessels and which results in the closure of one or more of such vessels in a given, circumscribed area. This leads to the creation of an ischemic zone and a consequent focal encephalomalacia. With the production of such cerebral softening, an area of diminished resistance is created which is an important if not a determining factor in the causation of cerebral apoplexy. In the presence of a diseased blood vessel and increased vascular tension, a reduction in the consistency of the surrounding tissue of the brain is an essential precursor to the rupture of the vessel wall and the unhindered escape of blood.

The anatomic manifestations in support of this view are: 1. The presence of generalized vessel disease. 2. The diffuse productive changes

throughout such brains. 3. The well defined organization in the wall limiting a hemorrhagic cavity. 5. The presence of exposed blood vessels in the hemorrhagic cavity. 6. The infrequent occurrence of miliary aneurysms in the group of cases we have studied.

DISCUSSION

Dr. E. W. Taylor, Boston:—If I understood Dr. Globus correctly, he maintains that primary cerebral hemorrhage practically never occurs. What importance has the original work of Bouchard and Charcot on the question of cerebral aneurysm? Charcot maintained that as a precursor of these cerebral hemorrhages, many aneurysms always occur. Whether or not such aneurysms are always the precursor of cerebral hemorrhage and whether in those cases we may have a hemorrhage irrespective of the area of softening are matters of interest.

Dr. Globus: In answer to Dr. Taylor's question, I would say that in hemorrhages of the so-called cerebral apoplexy there can always be found some evidence of a preexisting lesion. The miliary aneurysms of Charcot are only a part of the pathologic picture and are likely the result of two factors: the weakness of the vessel wall and the lack of support which is occasioned by the softening process in the vicinity of that vessel. It has already been shown by others that aneurysms are not common in cerebral hemorrhages and that cerebral hemorrhage may occur without the presence of such aneurysms. Hemorrhages without preexisting areas of softening do occur; they are, however, of a different type and are largely due to trauma.

With regard to the other question, I think that the difficulty lies in the fact that one commonly sees patients with cerebral hemorrhage in the acute stage, without having had the opportunity to study them at various times prior to the insult. Were such patients studied some time before the precipitation of the vascular explosion, it is likely that evidence of the preexisting cerebral lesion would have been found. It is a common experience in neurologic clinics to see a patient complaining of little more than headache, who on examination displays localizable signs pointing to the seat of a future cerebral hemorrhage.

THE ORIGIN OF MYELIN

A PRELIMINARY REPORT *

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My object in this study was to investigate the source or sources from which myelin is derived, the manner in which it is deposited around the axis cylinders in the central nervous system, and the nature and chemical evolution of its constituent elements. For the attainment of this object I was not primarily concerned with the chronologic schedule of myelinization in the different functional fiber systems, as this feature is being investigated by Tilney and Casamajor [1] in their "Studies of Myelinogeny as Applied to Behavior."

This problem was undertaken not merely for the purpose of ascertaining some isolated facts concerning this important constituent of the neurospinal axis but also of applying the information thus gained to that large group of diseases in which the failure of myelin to appear or its disappearance constitutes the outstanding pathologic features. Any new information concerning the source, organization and chemical constitution of myelin should have a fundamental bearing on that whole group of diseases characterized by amyelinogenesis, dysmyelinogenesis and myelinolysis.

The problem was approached with a tentative supposition that the deposition of myelin is a result of cellular activities. If that premise is true, it follows that it should be possible to discover an association between the cellular constituency of the axis at different periods of prenatal and postnatal development and myelinogeny. In order to investigate this, I determined to study the brains of rats at five different periods of development—prenatal, at birth, and at the ages of 1 day, 3 days and 8 days. The rat was selected because there is evidence that myelinization is almost wholly a postnatal activity. At least there appears to be little myelin in the nervous system when the animal is born. This statement is based on the fact that myelin, as such, does

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* Read at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 1, 1926.

* This research was made possible by a grant from the Commonwealth Fund for the investigation of multiple sclerosis. More information than is now possessed concerning the synthesis and dissolution of myelin is essential for a better understanding of this disease.

1. Tilney, Frederick; and Casamajor, Louis: Myelinogeny as Applied to the Study of Behavior, Arch. Neurol. & Psychiat. **12**:1 (July) 1924.

not show in the new-born rat under the application of the usual myelin stains. Paraffin, celloidin and frozen section methods were used, and a number of staining methods were employed.[2]

This report will be confined to a few observations that have been made in the first phase of the investigation. In the prenatal rat, observations have revealed small round cells scattered through the meninges that stain well with the Kulschitsky-Pal method. These cells do not differ in morphology or in the depth and color of staining from the contents of the blood vessels, but they differ sharply in their staining reaction from the few cellular elements in the nerve tissue that take the stain. The cells in the meninges and blood vessels stain deep bluish black, whereas the cellular elements of the axis stain brownish black.

The medulla, pons, midbrain, cerebrum and cerebellum are well outlined with these deeply bluish staining cells that appear to be located in the subarachnoid space (fig. 1). The ventricles also contain similar cells that are extravascular in location (fig. 8). With Weigert's hematoxylin and eosin stain, Delafield's hematoxylin and orange G stain, Mann's stain and toluidin blue, these are scarcely stained at all but are visible as skeleton or ring forms. At the periphery of the medulla, similar forms were seen beneath the pia and within the medullary substance of the axis (fig. 3).

Although myelin, in the condition ordinarily recognized in sections stained by the Weigert-Pal method, is not present in the prenatal rat, by varying the methods of decolorization and differentiation, diffusely

2. The brains of sixteen rats were sectioned serially and stained after paraffin embedding. Six staining methods were used: (1) Delafield's hematoxylin and eosin; (2) Weigert's hematoxylin and orange G; (3) Mann's stain; (4) toluidin blue; (5) osmic acid, 1 per cent, and (6) Busch's modification of the Marchi method. Fixation in solution of formaldehyde was used for methods 1 and 2, Bouin's fluid for 3, corrosive sublimate for 4, osmic acid for 5, and Müller's fluid followed by Busch's fluid for 6.

The brains of eight rats were fixed in Müller's fluid, embedded in celloidin, and serially sectioned at 50 microns, mordanted in 5 per cent copper bichromate, stained with Kulschitsky's hematoxylin and differentiated as for the Weigert-Pal method.

The brains of sixty rats were sectioned with the freezing microtome and were treated as follows: (1) scharlach R-hematoxylin; (2) the same after acetone; (3) the same after alcohol; (4) the same after chloroform; (5) zinc acetate mordant, scharlach R-hematoxylin; (6) zinc acetate mordant, alcohol, scharlach R-hematoxylin; (7) zinc acetate mordant, methyl alcohol and chloroform, scharlach R-hematoxylin; (8) zinc acetate, acidulated alcohol and ether, scharlach R-hematoxylin; (9) 1 per cent osmic acid direct; (10) osmic acid after chromication (Marchi); (11) Anderson's modification of the Kulschitsky-Pal method; (12) Nile blue sulphate; (13) neutral red; (14) Bielschowski method; (15) Hortega's stain for oligodendroglia; (16) Hortega's stain for migroglia; (17) Hortega's glia stain; (18) Cajal's gold sublimate method; (19) hematoxylin after copper acetate mordant, and (20) toluidin blue.

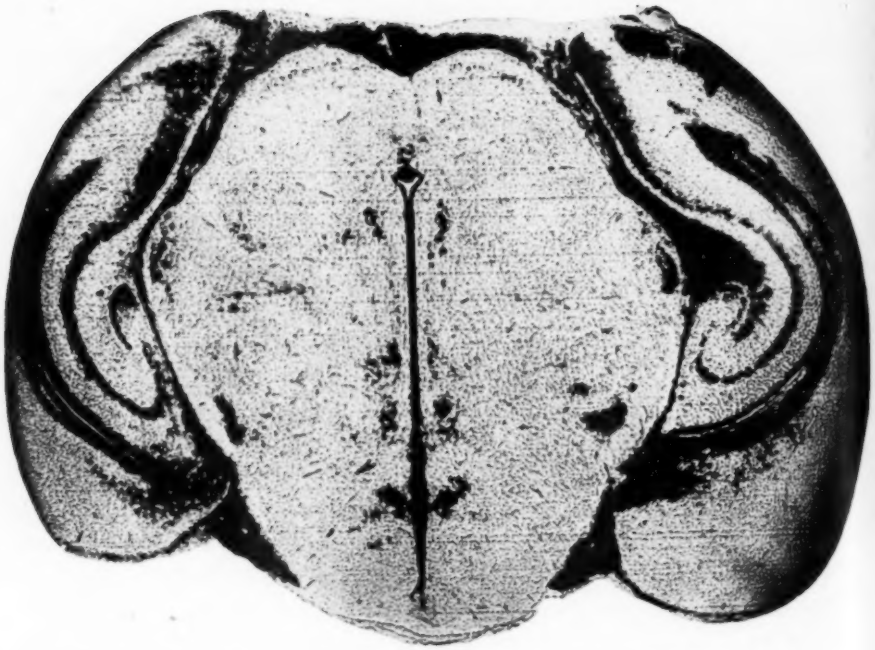

Fig. 1.—Prenatal rat. Section through the metencephalon showing an abundance of cells in the spaces of the pia-arachnoid covering the brain stem and cerebellum that appear black in the picture. Kulschitsky-Pal stain.

Fig. 2.—Prenatal rat. Section through the metencephalon showing globular forms in the meninges and periphery of the axis having the morphology and staining reaction of red blood cells. Kulschitsky-Pal stain. × 325.

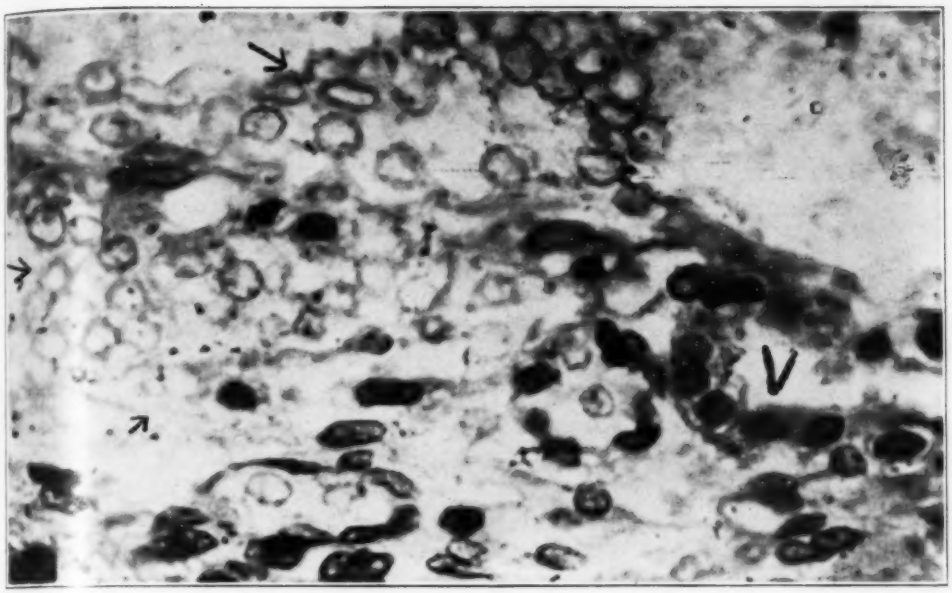

Fig. 3.—Prenatal rat. Section through the myelencephalon showing unstained globular forms in the meninges and marginal zone of the axis indicated by arrows. The lateral process of the fourth ventricle may be seen on the right of the picture at *V*. Weigert's hematoxylin and eosin. × 800.

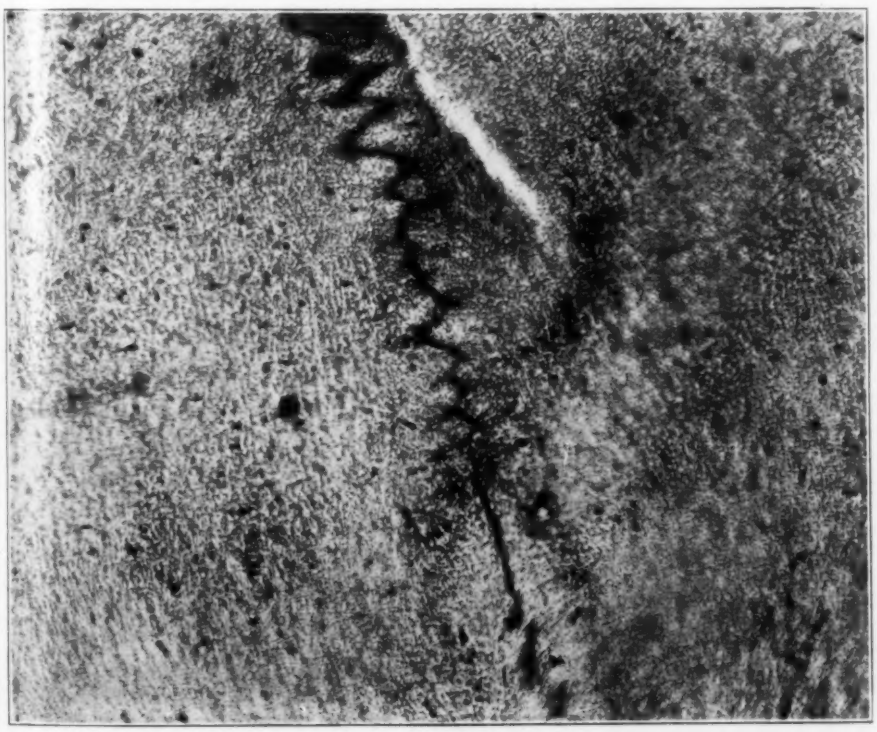

Fig. 4.—Prenatal rat showing the raphe stained. Kulschitsky-Pal stain (Anderson modification).

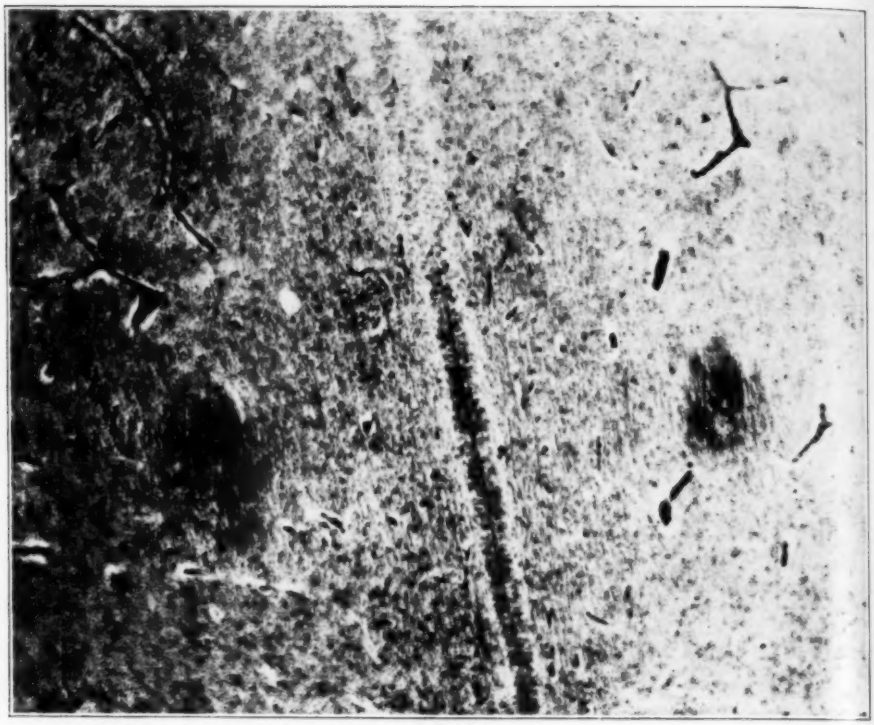

Fig. 5.—Rat at birth showing bilaterally and symmetrically situated longitudinal bundles in the metencephalon that stain like myelin but are lacking in the usual structural appearance of that substance. A homogeneity and lack of definition are present. Kulschitsky-Pal stain (Anderson modification).

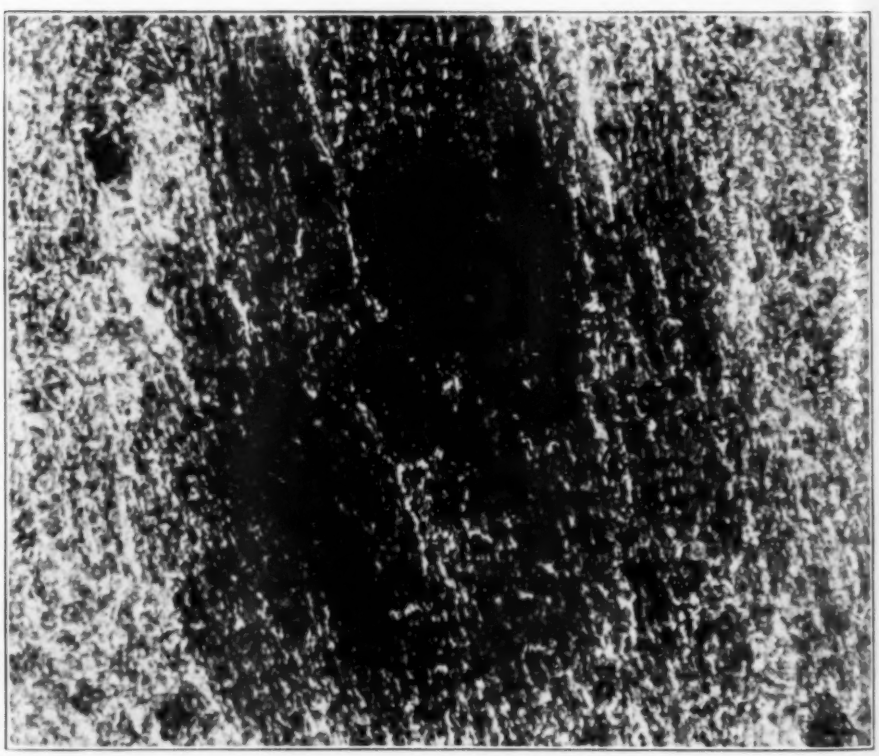

Fig. 6.—Rat at birth. Higher magnification of one of the bundles shown in figure 5.

stained cloudy areas may be seen. One area of this kind was observed in several animals in a constant location just lateral to the sylvian aqueduct (fig. 5). It was thought to represent a longitudinal bundle, most probably the fillet. Under a high power magnification fibrils were not seen; only a dark homogeneous mass (fig. 6) was visible.

There is little differentiation of the cells of the axis at this period. The cells of the inferior olive are small and round, about the size of lymphocytes, and possess almost no cytoplasm. The cells of the nucleus hypoglossus possess scarcely more than a ring of cytoplasm surrounding

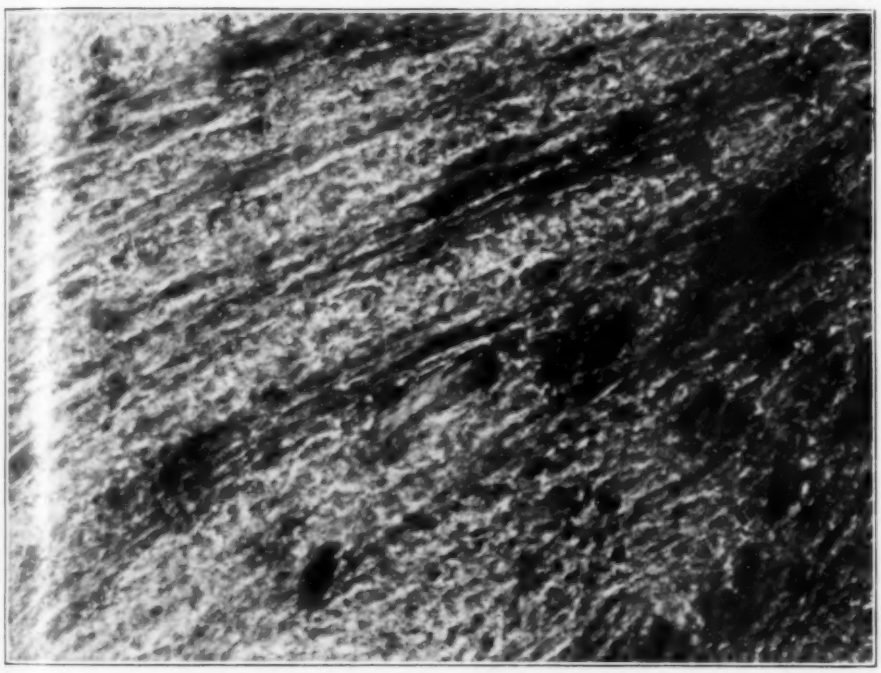

Fig. 7.—Rat at birth. Section through the ventral portion of the pons showing tubular structures that stained faintly with lithium hematoxylin after preliminary mordanting in copper acetate. This staining reaction indicates that they contain fatty acids.

the nucleus, and the nuclear chromatin is small in amount and delicate in structure.

When the scharlach R-hematoxylin stain was used after zinc acetate (Ciaccio's method) and also with lithium hematoxylin after copper acetate (Klotz' method), some sheath-like structures resembling hollow tubes could be distinguished. These stained faintly blue with hematoxylin, and this staining reaction is believed to be due to fatty acids (fig. 7). With the Anderson modification of the Kulschitsky-Pal

method for frozen sections, the raphe was stained black and was represented by a serpiginous line (fig. 4).

At birth, a diffuse cloudiness was noted in the parts of the medulla in which myelin first appears, that is, along the median raphe in the position later to be occupied by the posterior longitudinal and predorsal fasciculi and along the paths of the fifth, eighth and twelfth nerves. In the vicinity of the descending fifth nerve, the restiform body and the pars basalis of the pons, an abundance of small, round, deeply stained cells in dense clusters was found. At later stages, when myelin had

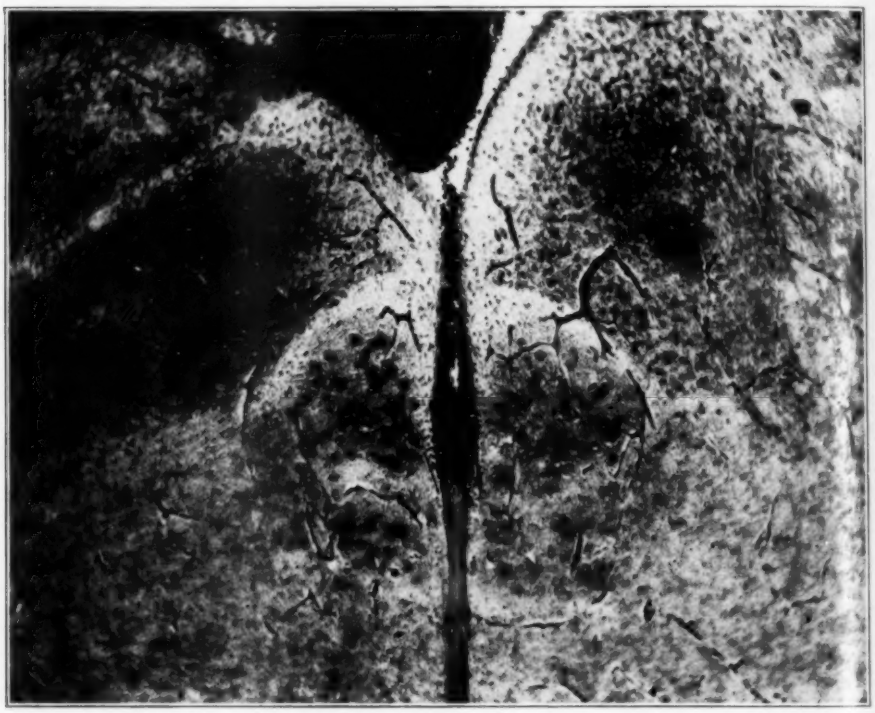

Fig. 8.—Rat, aged 1 day. Section at the level of the nucleus hypoglossus, showing an abundance of capillaries within and around this nucleus at the time that the twelfth nerve fibers are beginning to myelinize. The ventricle is filled with globular forms similar to those seen in the meninges. These appear to be entering the axis along the raphe. Kulschitsky-Pal stain (Anderson modification).

become more abundant, these clusters disappeared. This clustering of cells preceding myelinization has been noted by Tilney and Casamajor.[1]

At this stage, what was interpreted as hypervascularity of the axis was also observed, especially in the medulla oblongata. The vascular elements seemed to be more abundant in the regions soon to be occupied by myelinized fibers of the eighth and twelfth nerves. At the level of

the nucleus hypoglossus, the vascular radicles appeared to converge on this nucleus (fig. 8). Outside some of the capillaries a few small, round and oval globular masses that stained similarly to the contents of the capillaries could be seen. These masses suggest vascular extrusions. They were smaller than any of the cellular elements in the axis.

With the scharlach R-hematoxylin stain used after zinc acetate, lithium hematoxylin after copper acetate, and 1 per cent osmic acid, the same sheath-like structures that were observed in the prenatal animal were seen. This appearance was especially noticeable in the ventral

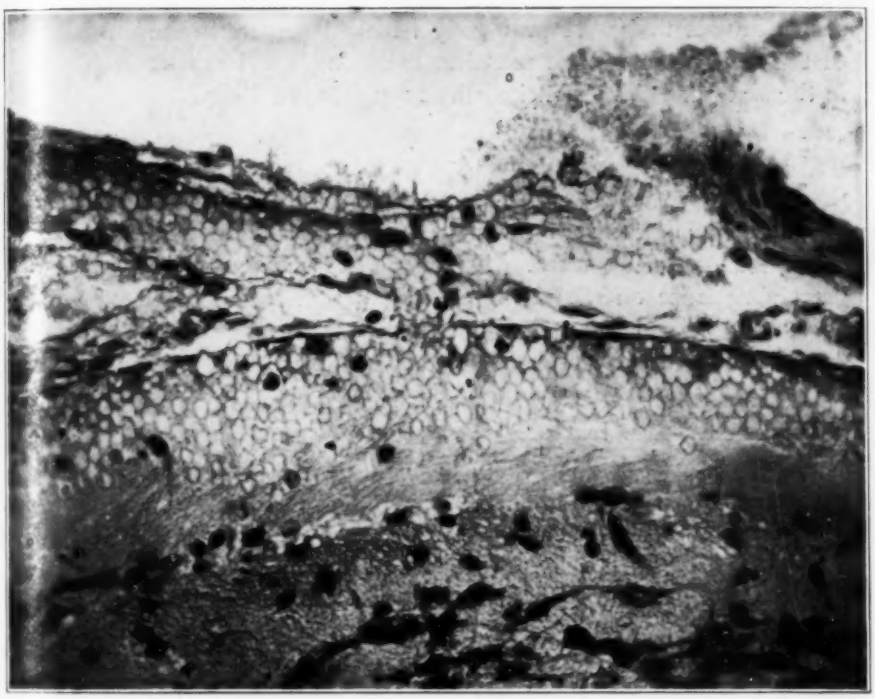

Fig. 9.—Rat, aged 1 day. Section through the medulla. A blood space in the pia-arachnoid appears to be discharging its contents directly into the periphery of the neuraxis. Weigert's hematoxylin and eosin. Reduced from a photomicrograph with a magnification of 800 diameters.

portion of the pons on each side of the median raphe, where they were disposed in symmetrical curves with the convexities upward (fig. 7).

In the lateral processes of the fourth ventricle and in the subarachnoid space between the medulla oblongata and the cerebellum, near the distal extremities of these processes, dense groups of cells were present. These were similar to those seen in the same localities in the prenatal animals. At the periphery of the medulla oblongata, in a dorsolateral situation, large collections of the same cells were present

beneath the pia. One day after birth, these cells were still more prominent, and in one of the brains that was serially sectioned and stained with Weigert's hematoxylin and eosin these cells appeared to be passing from a blood channel of the pia-archnoid into the medullary substance (fig. 9). Coincident with this, myelin appeared in the descending fifth, twelfth and eighth nerves, the corpus trapezoideus, the mesencephalic fifth and the fasciculus longitudinalis posterior. At the same time, the neurocytes of the axis became more differentiated. The nuclear chromatin was more abundant and coarse, and the cytoplasm was increased in amount and contained some tigroid substance. The same features that were previously noted in respect to the vascularity of the eighth and twelfth nuclei in the animals at birth were observed in the sixth and third nuclei of the animals, aged 1 day.

SUMMARY

The following observations cover the first step in this investigation:

1. Before myelinization occurs, the vascularity of the neuraxis is richer than at later periods. The blood vessels appear to converge in great abundance on the nuclei of the twelfth, eighth, sixth and third nerves before and simultaneously with the first appearance of myelin around the axons of these nerves.

2. Preceding myelinization, cells are present in the ventricles and subarachnoid space that are extravascular in location and that stain similarly to the contents of the blood vessels with myelin stains.

3. At birth and at 1 day, cells having an appearance similar to that of the cells in the ventricles and subarachnoid space may be seen within the axis in a peripheral situation at the level of the lateral processes of the fourth ventricle.

4. Structures having the appearance of hollow tubes or sheaths may be seen in the axis before myelin appears. The staining reactions of these tubular structures would indicate that they contain fatty acids.

A SYNDROME OF THE CEREBRAL ORIGINS OF THE VISCERAL NERVOUS SYSTEM

THE QUESTION OF SOMATIC AND VISCERAL ATROPHY AND A REVIEW OF THE REPORTED CASES OF UNILATERAL ATROPHY *

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The unusual combination of progressive growth resulting in gigantism and mild acromegaly, of muscular atrophy of the extremities and trunk on one side of the body resulting in hemiparesis and associated with creatinuria and absence of reaction of degeneration, of mild hypersomnia and of diabetes insipidus which was controlled by the administration of solution of pituitary is the reason for reporting the following case.

It is our belief that all of these manifestations have their origin in the cerebral visceral parts of the central nervous system which control the physicochemical activities of the body and are located in or about the floor of the third ventricle.

Recently, renewed interest has been taken in this portion of the nervous system, and evidence has accumulated to show that the cell groups lying about the tuber cinereum play an important part in regulating the sleep cycle, temperature, the metabolism of fats, sugar, salt and water, as well as the viscera, including the glands of internal secretion. The accumulated evidence up to the present leads us to believe that these cerebral portions of the visceral nervous system exert a trophic control not only on the glands and smooth muscle but also on striated muscle, tendons, bones, skin and hair, and, indeed, on all the tissues of the body.

The difficulties of the subject are, in the main, three: (1) The pituitary gland lies in close relation anatomically to the floor of the third ventricle, and as a consequence it has not been easy to state whether certain signs and symptoms are of pituitary origin or arise from injury of the adjacent nervous system. (2) The cellular masses in this region are not extensive or numerous, and it is hard to understand how these small cell masses can control so extensive and varied a group of activi-

* Read by title at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1926.

ties. (3) A number of diseases show some but not all of the signs and symptoms that could be attributed to this area though it is hard to say why. The detailed case report shows that this patient had progressive wasting of the muscles of the trunk and extremities of the right side, beginning at the age of 23 and progressing slowly over a period of two years and three months.

It is interesting that a large number of partial hemiatrophies affecting the face and sparing the limbs and trunk of the same side are on record, while the reverse condition, such as is presented by our case, is not reported, as far as we have been able to find. Pelizaeus, in 1897, reported the case of a child who showed unilateral atrophy of the limbs and trunk. The face was not affected. The atrophy appeared at the age of 4, soon after a fall. In many respects this case differs from ours, and we do not believe that it belongs in precisely the same category.

Beyond slight pain in the arm while pitching a baseball, which called attention to the wasting, our patient had had no sensory disorders whatever. Prior to the onset, he was an extremely powerful young man, had been active in athletics, and had followed the strenuous occupation of furniture mover.

The wasting is demonstrated in figures 1 to 6. Figure 6 shows the sign of Tournay and Kraus,[1] which has previously been described in isolated paralysis of the serratus magnus. It is our opinion that the presence of this sign here is dependent on the weakness of the serratus rather than on that of the trapezius. The muscles of mastication supplied by the fifth nerve and the muscles of facial expression supplied by the seventh nerve are not affected, beyond slight ptosis.

The electrical reactions in this case are also interesting in that the patient did not show any reaction of degeneration, but merely a change in response to the galvanic current which was manifested as ACC. > CCC. There is a great difference between the electrical reactions of somatic and of visceral atrophy, and we wonder whether the inversion of the poles in our case may be considered as a manifestation of a disorder of the physicochemical state of muscle tissue in contrast to a disorder affecting the impulses that govern its movements.

It has long been felt that muscular dystrophy had its origin in a disorder of the central nervous system, but no conclusive data to prove this have yet appeared.

It seems reasonable to believe that a great difference exists in the type of muscular atrophy, depending on whether this is due to a lack of the impulses that make muscles move or to a lack of impulses that main-

1. Tournay, A., and Kraus, W. M.: J. Neurol & Psychopath. **5**:115, 1924-1925.

tain their trophic control; that is, their normal physicochemical state. Regarded from this point of view, what is now spoken of as secondary or myelogenic atrophy becomes identical with what we would call "somatic muscular atrophy," and what is now spoken of as primary muscular atrophy or muscular dystrophy we would call "visceral muscular atrophy." We do not mean to imply by this that the causative lesion of muscular dystrophy is always in the cerebral portion of the

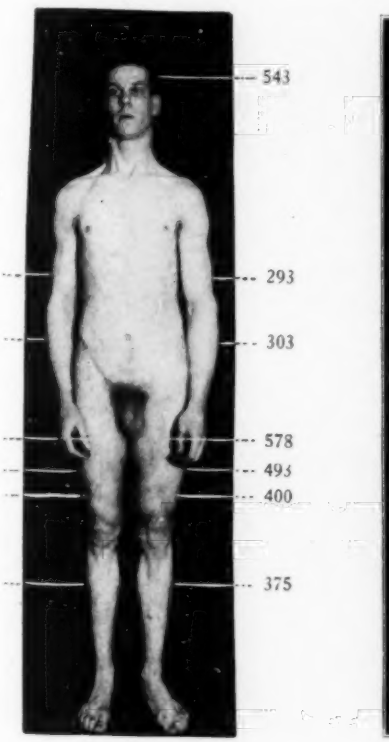

Fig. 1

Fig. 2

Fig. 1.—Atrophy of right side, except the face, gigantism, and normal genitals. Circumferences in numbers indicate millimeters.

Fig. 2.—Rear view of the same patient as in figure 1, which also shows the wasting about the right shoulder.

vegetative nervous system. It may affect any part of the pathway that runs between these central origins and the muscles themselves. On the other hand, somatic muscular atrophy, as far as we know, is always due to a lesion of the terminal neuron of the system which make muscles move; that is, the final common pathway of Sherrington, the anterior horn cell and its axon.

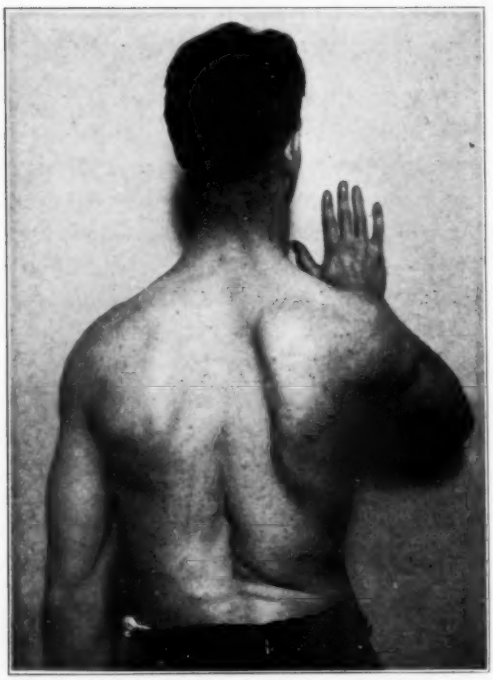

Fig. 3.—Winging of the right scapula due to wasting.

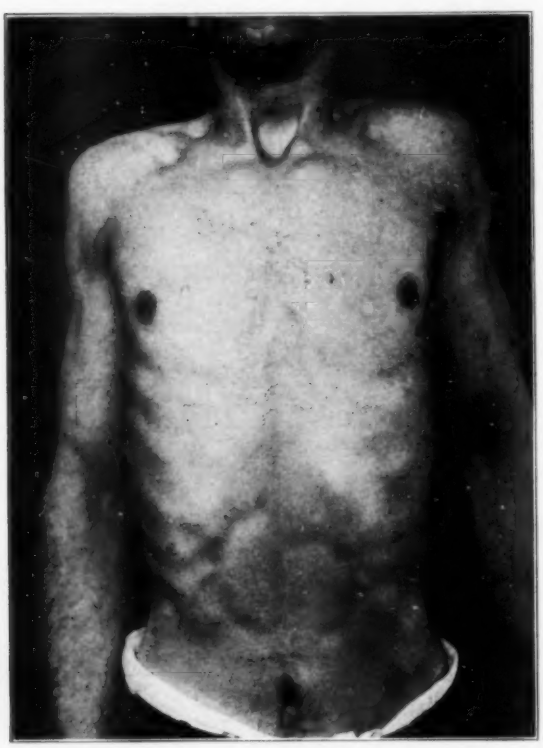

Fig. 4.—Wasting of the intercostals on the right side.

It is of importance in this connection that visceral atrophy (dystrophy) is never associated with fibrillary twitchings, while somatic atrophy always is so associated at some stage of its development.

The double supply of muscles by the sympathetic and somatic nervous system is well established morphologically, as far as the spinomuscular neurons are concerned, while the question of the physiologic aspects of this double supply is still unsolved.

Tempting as it may be to carry the question of somatic and visceral atrophy over into that of the double innervation of muscles, it seems unwise at present to suggest the precise pathways which have to do with the types of atrophy, somatic and visceral, which have been defined.

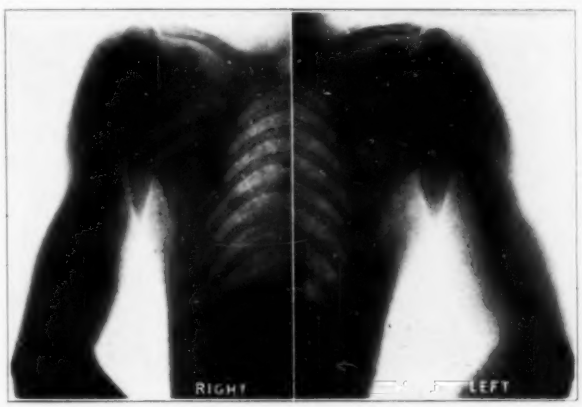

Fig. 5.—Wasting of the soft parts of the right arm, which may be easily measured through the belly of the biceps.

GIGANTISM AND ACROMEGALY

The close relation of tumor of the pituitary gland to the two bony disorders leading to acromegaly and gigantism has been well established by the work of Brissaud, Marie, Meige, Launois and Roy, Beidl and Cushing. In addition, trauma, infection and inherited characteristics are among the less frequent causes.

It has also been shown that the syndromes that seem to be of pituitary origin may be unassociated with diseases of the pituitary gland and may be closely associated with disorders of the region of the tuber cinereum. Per contra, lesions of the pituitary gland are not always associated with what are commonly regarded as pituitary symptoms.

Recently, Greving [2] has shown that there is a pathway between the nucleus supra-opticus in the tuber cinereum region and the pituitary

2. Greving, R.: Beiträge zur Anatomie des Zwischengehirns und seiner Funktion, Ztschr. f. d. ges. Neurol. u. Psychiat. **99**:231, 1925.

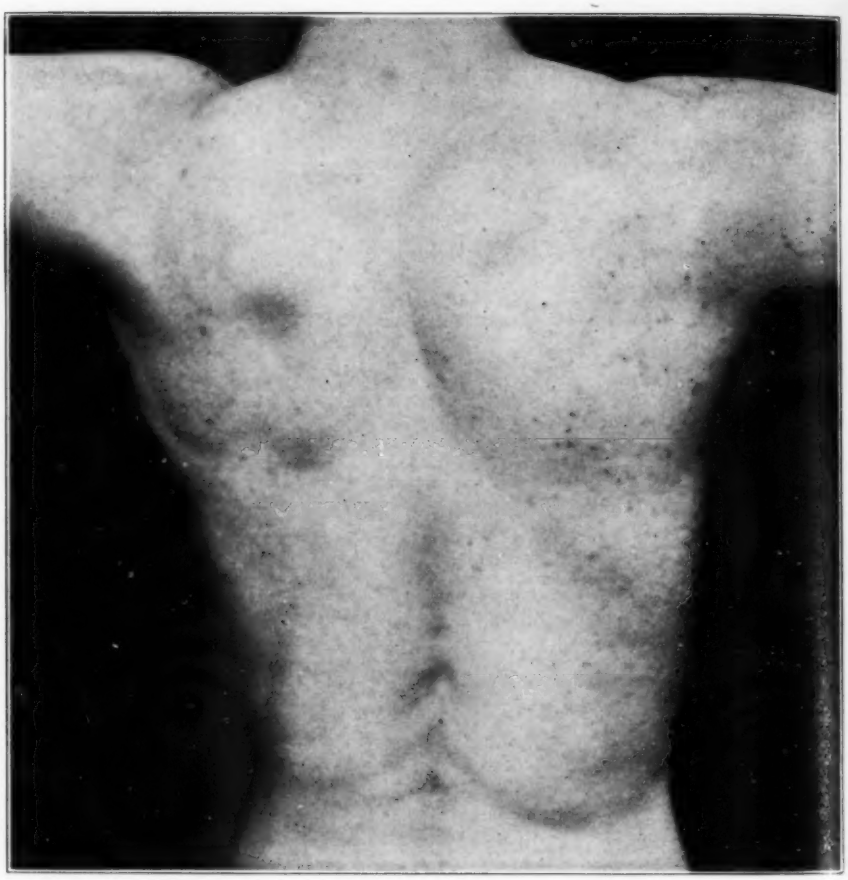

Fig. 6a

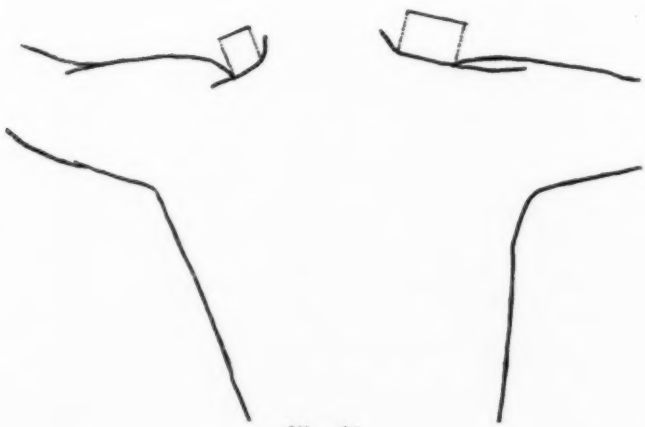

Fig. 6b

Fig. 6.—The sign described by Tournay and Kraus in unilateral paralysis of the serratus magnus.

gland itself. In our case, the roentgen-ray examination of the sella turcica and the neurologic examination, including that of the visual fields, have not given any evidence that the pituitary gland is diseased.

If we had no other manifestations referable to the floor of the third ventricle, we would hesitate to state that the bony changes in our case were primarily due to disorders of the nervous system. As it is, we feel that this is the best explanation that can be given.

In a recent paper, Roussy [3] has expressed the opinion that the bony changes of what, in the past, has been called a pituitary syndrome are always due to disorders of this gland. In view of the observations in this case, we question whether so sweeping an opinion is as yet justified. Certainly, changes in the bones and joints due to spinal disease are well known, e. g., tabes dorsalis, syringomyelia and transverse lesions of the cord.

Whether, in some cases, the bony changes of acromegaly and gigantism are produced by disorders of pathways between the floor of the third ventricle and the pituitary gland, or by disorders of the pathway going caudally from the floor of the third ventricle without passing through the pituitary gland and affecting primarily the trophic control of the bones is a question that must be decided in the future.

WATER METABOLISM

It has been abundantly shown that the region of the tuber cinereum controls water metabolism. It is apparent that a tumor pressing on this region may produce the same result. The mild grade of diabetes insipidus that our patient has shown is an added bit of evidence of involvement of this part of the nervous system. That it has been controlled by the administration of pituitary extracts in our case falls in line with clinical experience.

CREATINE

Creatine secretion on a meat-free diet was extremely high, 0.4 Gm. In normal males creatine is not present Its presence indicates a disorder of the chemical activities of the muscles.

HYPERSOMNIA

The mild grade of hypersomnia that this patient showed has not persisted since the administration of the solution of pituitary. Hypersomnia has been reported in tumors of the pituitary gland as a symptom most probably referable to the region of the tuber cinereum and the upper midbrain. This observation serves as another link in the chain of evidence that we are presenting.

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3. Roussy, Gustav: Les fonctions de la région infundibulo-tubérienne et ses rapports avec l'hypophyse, Ann. de méd. **18**:407, 1925.

UNILATERAL ATROPHY

Unilateral atrophy, unassociated with motor disorders such as unilateral paralysis and unilateral athetosis, is extremely rare, while the cases that are associated with such motor disorders are common. Infantile and adult unilateral paralysis are often associated with

TABLE 1.—*Reported Cases of Unilateral Atrophy*

Year	Author	Sex*	Side	Age at Onset	Mental Defect	Remarks
			Group 1 B.—Congenital Group			
1859	Broca, Paul (Canstatt's Jahresbericht **4**:26)	♂	Right	Congenital	None	
1883	Pixley, S. (M. News **43**: 146)	♂	Right	Congenital	None	
1884	Burrell, H. L. (Boston M. & S. J. **3**: 462)	♀	Left	9	None	
		♀	Left	Age of walking, 1½	None	Legs only affected
		♂	Right	Age of walking, 1½	None	Legs only affected
		♀	Left	Age of walking, 1½	None	Legs and face affected
1911	Geist (Neurol. Centralbl. **29**: 122)	♂	Left	Congenital	+	
1923	Léri, A., and Tzanck (Bull. Soc. franç. de dermat. et syph. **30**:210)	♂	Left	Congenital	?	Hemihypotonia, hemioculosympathetic syndrome, hemisweating, neurofibromatosis, nevi
			Group 2.—Familial Group			
1902	Raymond and Sicard (Rev. neurol. **10**: 593)	♀	Left	20	None	Ascending type
		♂	Left	17	None	Descending type
		♀	Right	20	None	Ascending type
			Group 3 B.—Acquired Group			
1896	Luntz (Neurol. Centralbl. **15**: 1045)	♀	Right	22	None	Began in face on one side and affected limbs on other later, crossed type
1897	Pelizaeus (Neurol. Centralbl. **16**: 530)	♀	Left	Noted at 4, after fall	None	Face not involved
1903	Debray, A. (J. de neurol. **8**: 63)	♂	Left	18	None	Ascending type; facioscapulo-humeral type
1908	Orbison (J. Nerv. & Ment. Dis. **35**: 695)	♂	Left	18	None	
1913	Bonnet and Gaté (Lyon méd. **121**: 793)	♀	Left	16	None	Acquired syphilis
			Group 4.—Not Classified			
1910	Meyer, E. (Neurol. Centralbl. **29**: 450)	♂	Left	? Not congenital	+	Hemiparesis (may belong in group 3 A)
1923	Hajashi,, M. (Neurol. Centralbl. **35**: 275)	?	?	?	?	Brain atrophy; pathologic report only

* In this column, ♂ indicates male, and ♀, female.

unilateral atrophy. Table 1 gives a summary of the eighteen reported cases of simple unilateral atrophy.

For purposes of classification we propose the following division of the reported cases. For completeness we have included those groups associated with motor disorders to which we have just alluded, with a few examples.

1. Congenital:
   (*a*) Associated with unilateral paralysis and unilateral athetosis. Weber's [4] and De Boyer's [5] cases are examples.
   (*b*) Unassociated with unilateral paralysis or unilateral athetosis, eight cases—Broca,[6] Pixley,[7] Burrel;[8] four cases—Geist,[9] Léri and Tzanck.[10]
2. Familial: Three cases of Raymond and Sicard.[11]
3. Acquired:
   (*a*) Associated with unilateral paralysis and unilateral athetosis, Example: Taylor's case.[12]
   (*b*) Unassociated with unilateral paralysis and unilateral athetosis, five cases—Luntz,[13] Pelizaeus,[14] Debray,[15] Orbison,[16] Bonnet and Gaté.[17]
4. Not classified, cases of Hajashi [18] and Meyer.[19]

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4. Weber, F. P.: Right-Sided Hemi-Hypotrophy Resulting from Right-Sided Congenital Spastic Hemiplegia, with a Morbid Condition of the Left Side of the Brain, Revealed by Radiogram, J. Neurol. & Psychopath. **3**:134, 1922.

5. De Boyer, H. C.: Atrophie cérébrale et cérébelleuse croissée. Asymétrie de la moelle. Atrophie du pied de la frontale et de la pariétale ascendante, chez un sujet atteint depuis son enfance d'atrophie des membres et de contracture à forme hémiplegique, Progrès méd., 1878, pp. 142-143.

6. Broca, Paul: Angeboren Asymmetrie der beiden Körperhälften, Canstatt's Jahresbericht **4**:6, 1859.

7. Pixley, S.: Congenital Unilateral Atrophy, M. News **43**:146, 1883.

8. Burrell, H. L.: Unilateral Atrophy, Boston M. & S. J. **3**:462, 1884.

9. Geist: Ein Fall von halbseitiger Unterentwicklung, Neurol. Centralbl. **29**:122, 1911.

10. Léri, A., and Tzanck: Naevi verruqueuse et lésions nerveuses multiples. Maladie de Recklinghausen fruste, hémiatrophie, hémihypotonie, hémisudation, hémisyndrome oculosympathique, Bull. Soc. franç. de dermat. et syph. **30**:210, 1923.

11. Raymond and Sicard, J.: Trophonévrose hémiatrophique totale et familiale, Rev. neurol. **10**:593, 1902.

12. Taylor, T.: Unilateral Atrophy with Muscular Spasm, Lancet **1**:387, 1878.

13. Luntz, M. A.: Hemiatrophia Totalis Cruciata. Gesellschaft der Neuropathologen und Irrenärzte zu Moskau, Jan. 19, 1896; Neurol. Centralbl. **15**:1045, 1896.

14. Pelizaeus: Ueber einen ungewöhnlichen Fall von progressiver Hemiatrophie, Myosclerose, Sclerodemie und Atrophie der Knochen und Gelenke, Neurol. Centralbl. **16**:530, 1897.

15. Debray, A.: Hémiatrophie facio-scapulo-humérale, J. de neurol. **8**:63, 1903.

16. Orbison: Trophic Complete Hemiatrophy. A Trophoneurosis, J. Nerv. & Ment. Dis. **35**:695, 1908.

17. Bonnet, L. M., and Gaté, J.: Hémiatrophie de la moitié gauche du corps, Lyon méd. **121**:793, 1913.

18. Hajashi, M. Ueber cerebrale Hemiatrophie. Jahresversammlg. d. Deutsch. Vereins f. Psychiatrie, Neurol. Centralbl. **35**:275, 1923.

19. Meyer, E.: Totale Hemiatrophie, Neurol. Centralbl. **29**:450, 1910.

We have found reports of only eighteen cases without motor disorders. The present case constitutes the nineteenth.

The atrophy does not always affect the entire right or left side. The following varieties have been reported: (1) facial, abundantly reported (Romberg); (2) facioscapulohumeral (Debray); (3) crossed, face on one side, limbs and trunk on the other side (Luntz); (4) infrafacial type (Pelizaeus and the case reported here); (5) crural type (Burrell); (6) complete (Broca, Pixley, Geist, Léri and Tzanck, Raymond and Sicard, Orbison, Bonnet and Gaté, Hajashi and Meyer).

The cases may also be classified according to the manner in which they progress. The entire side may be affected at once or the disease may be ascending or descending. The varieties indicate either that parts of paths from the region of the tuber, or parts of the nuclei of origin are affected. The distribution among the sexes is equally divided. The distribution as to right or left sides is: right, five, and left, twelve; one unknown.

Mental defect was noted in two cases only (Geist and Meyer). In fourteen cases none was present, and in the remainder no statement was made. In this connection, it is interesting that in unilateral hypertrophy, a condition which, although rare,[20] has been reported much more frequently than unilateral atrophy, 13 per cent of the cases show mental defect. Another contrast between the two groups of disorders is that the hypertrophies are frequently associated with skin disorders of a congenital character, while only one of the eighteen cases of atrophy shows this. The ages of onset in the familial and acquired groups were 20, 17, 20, 22, 18, 19, 16 and 4. The last case was of traumatic origin. The electrical responses were reported in six cases and consisted of lowered response to the two currents, without any reaction of degeneration, a type of response characteristic of visceral muscular atrophy (dystrophy). Shortening of the leg on the affected side occurred only in those cases which developed before puberty. No etiology could be assigned in any of these cases, with the possible exception of trauma reported in the case of Pelizaeus (onset at the age of 4), nor were any of them hereditary.

ETIOLOGY

In our case there is no evidence that the condition is hereditary, congenital or familial. There are neither skin lesions nor mental defects. There is no evidence of disease of the cortex or basal ganglia, nor is there any definite evidence of trauma, neoplasm or infection.

REPORT OF CASE

History.—E. L., a man, aged 24, born in the United States, came to the Neurological Clinic, Long Island College Hospital, because of weakness of the right arm. The mother of the patient was born in Sweden. She was the oldest

20. Gessell, A.: Hemihypertrophy and Mental Defect, Arch. Neurol. & Psychiat. **6**:400 (Oct.) 1921.

of a family of five, three girls and two boys, all living. Only one member of this group is tall, about 6 feet. The father was born in Sweden and had three brothers and three sisters. Nothing physically abnormal could be determined in this family. There is no history suggesting nervous or endocrine disturbances in either the maternal or the paternal families. The patient has one sister, living, who has been examined by both of us. She is 30 years of age, is 5 feet 8 inches tall, is healthy and does not show any abnormalities. One sister died at the age of 4 from diphtheria.

The patient was a full term infant, normally delivered, with a short labor. He had measles and whooping cough in infancy. Since the age of 5, he has been healthy and free of illness of any type. He began school at the age of 6 and continued up to the age of 14. He made good progress in school and took an active parts in sports, especially track and baseball. He was right-handed. He became a baseball pitcher while at school and continued to pitch in semiprofessional baseball up to the age of 22. He worked as a bank messenger after leaving school. Later, he worked on a ranch in Arizona for three years. Up to the present he had been a furniture mover.

History of the Present Complaint.—He dates the onset of the present complaint from September, 1924. While pitching in a game of baseball, he noticed that it required more effort and that he was unable to throw the ball with as much speed as in previous games. During the game his arm became weaker, and he noticed twinges of pain in the muscles of the right arm only. Following this experience, the right arm became progressively weaker and has continued to do so up to the present time. Six months after the onset in the arm, he noticed that the right leg was becoming weak and this has also been progressive. There has not been any tremor of the hand or fingers, and he has never noticed any twitchings in the muscles of either arm or leg. He can accomplish fine and complicated movements with the fingers of this hand as well as ever, if the movements are not continued over too long a period of time, otherwise the fingers and arm become tired and weak. He has not experienced any sensations of "pins and needles," numbness or pain in these parts. He has realized that the right arm and leg are smaller than the left.

He has increased 2 inches in height in the past two years. In 1924, he wore a hat, size 6½; gloves, size 8; shoes, size 8, and collar, size, 14. At present he wears a hat, size 6⅞; gloves, size 11; shoes, size 9½ and collar, size 15.

He requires more sleep at present than heretofore, although he is not sleepy during the day. For the past two years he has had a constant sense of thirst and has drunk more water than previously. He urinates twice at night and from five to eight times during the day. He has also developed a fondness for sweets during this time.

In December, 1924, he fainted while at work. He was unconscious for about one-half hour. On regaining consciousness he felt weak and dizzy but had no headache. This cleared up in about an hour, and he continued work. Eight months later, he had a second fainting spell which was similar to the first. He has not had any since. He was not seen in either of the two attacks, and they have not persisted; consequently, we do not feel justified in commenting on them. The patient has never had headaches or any other symptoms of increased intracranial pressure.

Examination.—The patient was well nourished, with good muscular development. There were no abnormal attitudes or deformities. The gait was normal, with associated movements of trunks, arms and head. With the feet together, he stands equally well with the eyes open or closed. There is no evidence of

ataxia, dysmetria or dyssynergia, and there are no tremors, twitchings, choreiform movements or muscular spasms. The deep reflexes of the right upper and lower extremities are diminished by comparison with those of the left. No pathologic reflexes are present. The superficial reflexes are present and equal.

There is a generalized weakness of the flexors and extensors of the right arm and leg as compared with the left. There is marked atrophy of the right arm and leg as compared with the left. Comparative measurements are found in figure 1. There are normal consistency, tone and absence of tenderness in the muscles of both sides. In testing the electrical reactions, there was no reaction of degeneration, but a change in the galvanic current, which was manifested as A C C. > C C C. All tests for touch, pain, temperature, vibratory, muscle and tendon sense elicit prompt and accurate responses.

Cranial Nerves.—The first nerve was normal. The second, third, fourth and sixth showed normal vision 20/20 in each eye; the fields were normal for form and color; the fundi were normal; the pupils were equal and reacted in accommodation and to light; slight ptosis of the right eyelid was present; all extraocular movements were equal and full; no nystagmus, enophthalmus or exophthalmus was present. The fifth nerve showed: muscles of mastication strong and equal, and tests for sensation normal; the seventh, no facial asymmetry or paralysis, and tests for taste negative; the eighth, no evidence of nerve deafness. The ninth and tenth were normal. In regard to the eleventh, although little difference was noted between the action of the sternocleidomastoids of the two sides, there was marked weakness of the right trapezius. The twelfth showed the tongue protruded in the midline. No atrophy was present.

Mental Condition.—There had been no change in the mental status of the patient.

Bodily State.—The patient was 1,856 mm. in height. The arms were equal in length, 787 mm., with a span of 1,850 mm. A roentgenogram of the chest taken to determine the presence of the thymus, was negative. Stereoscopic examination of the sella turcica and the skull showed that all accessory nasal sinuses were abnormally large; a typical acromegaly was present with prominent frontal ridge and malar eminences, and prognathism (fig. 7); the sella turcica was normal (fig. 8). Roentgen-ray examination of the pelvis showed that the hips, humeri, shoulders and feet were normal in bone development and that the epiphyseal lines were fused. On the pelvic roentgenogram was shown a definite, practically complete, sacralization of the fifth lumbar vertebra. The roentgenographic appearance of the upper arm on the left side was definitely denser than on the right (fig. 5). Examination of the feet revealed nothing abnormal.

Hair was abundant on the head and was distributed normally on the chest and extremities.

The genitalia were normally developed, and their function up to the present time had been normal. Examination of the heart, lungs and abdomen was negative. The electrocardiogram was normal.

Laboratory Examination.—The urine showed: specific gravity, 1.022; no albumin, glucose or casts; total twenty-four hour output, 1,950 cc. After forty-eight hours on a meat-free diet: creatinine, total 1.8 Gm.; creatinine, total 0.4 Gm. The blood showed: red cells, 3,540,000; white cells, 9,800; polymorphonuclears, 71; small lymphocytes, 22; large lymphocytes, 5; eosinophils, 1 per cent; hemoglobin, 80 per cent. Blood chemical determination (milligrams per hundred cubic centimeters) showed urea nitrogen, 15; urea, 32.10; uric acid, 2.27; creatinine, 1.50. Sugar tolerance: one and one-half hours after ingestion of 100 Gm. of glucose, the blood showed 118 mg. per hundred cubic centimeters. The blood Wassermann reaction was negative.

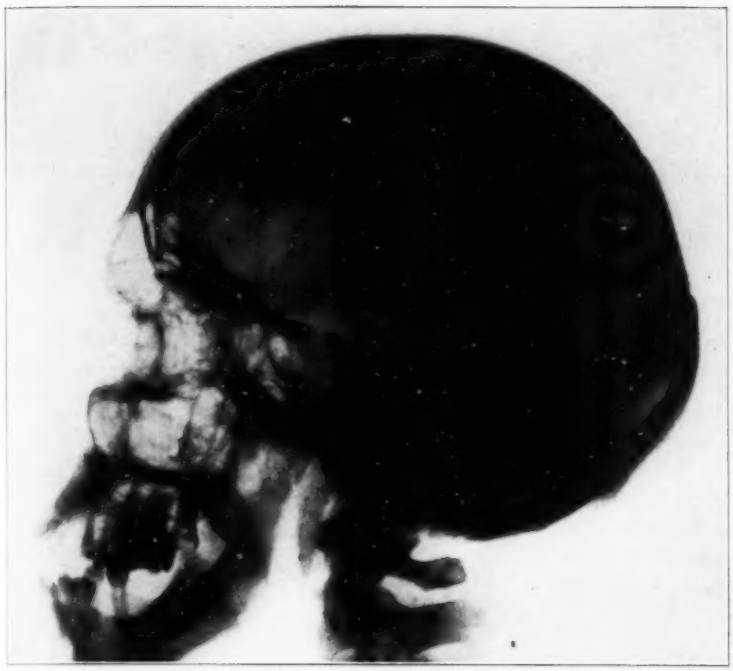

Fig. 7.—Acromegaly, as shown by prominent frontal ridge and malar eminences, and prognathism.

Fig. 8.—Normal sella turcica.

CONCLUSIONS

On the evidence of the case reported, we believe that it is possible to define another syndrome of the cerebral origins of the visceral nervous system.[21] We believe that the signs and symptoms of such syndromes may be extremely varied, but that they always include one or more signs or symptoms referable to the trophic or metabolic activities of the body.

We believe that this cerebral part of the central nervous system exerts a trophic control over muscles and bones and that the existence of this trophic control over muscles permits the description of the two recognized types of muscular wasting, atrophy and dystrophy, as somatic atrophy and visceral atrophy, respectively. The former is not related to disease of the visceral nervous system, while the latter is.

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21. Kraus, W. M.: Pilous Cerebral Adiposity; A New Syndrome, Am. J. M. Sc. **149**:737, 1915. (This is only one of many of these syndromes.)

THE CAJAL AND HORTEGA GLIA STAINING METHODS *

A NEW STEP IN THE PREPARATION OF FORMALDEHYDE-FIXED MATERIAL

J. H. GLOBUS, M.D.
NEW YORK

Formaldehyde is still the ideal fixation medium for the preservation of brain tissue. It hardens such material in bulk rapidly and uniformly; it is of great service in the study of changes in the central nervous system, for it offers an opportunity to retain the specimen in its uncut and undisturbed shape for gross morphologic studies before it is dismembered for finer cytologic observations. There is, however, one difficulty—and that is, because of the great assortment of methods devised for a better display of the various components of the nerve tissue and their numerous pathologic changes, there is need of a large variety of fixatives. This holds true for the many methods of staining neuroglia, and it is particularly essential for Cajal's [1] gold chloride and sublimate method as well as for its more recent modifications introduced by Achucarro and Del Rio Hortega.[2]

Cajal's staining method demands that fresh material be fixed in a formaldehyde-ammonium bromide mixture. Therefore, it is imperative for brains and spinal cords to be sectioned, soon after necropsy, into small blocks and immediately put into that mixture. Such a step precludes the study of such material in toto over longer periods or its employment for other staining methods in which fixation in ammonium bromide-formaldehyde is undesirable. It would be valuable to find a way that would allow thorough saturation with ammonium bromide of selected blocks or sections of the brain or the cord already hardened in formaldehyde.

With this in mind, a new step in the preparation of material for the valuable methods of Cajal and his students has been devised.

Fairly satisfactory results can be obtained by placing frozen sections of material previously fixed in formaldehyde into a 2 per cent aqueous

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* From the neuropathologic laboratory, Mt. Sinai Hospital.

* Aided by a grant from the Emanuel Libman fellowship fund.

* Read by title at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1926.

1. Cajal, Ramon y: Trab lab. biol. of the University of Madrid **11,** 1913; **14,** 1916.

2. Hortega, del Rio: Trab lab. biol. of the University of Madrid **15,** 1918; **16,** 1920.

solution of ammonium bromide, but success is not constant, the staining is not always uniform and such preparations are seldom available for photomicrography.

By utilizing frozen sections of nerve tissue as a vehicle for a chemical reaction, a more uniform and more complete saturation of the tissue elements with ammonium bromide can be obtained. Accordingly, frozen sections of desired thickness are placed in a suitable solution of ammonium hydroxide for a given length of time, and after rapid washing are acted on by hydrobromic acid. This apparently results in ample saturation of the tissue with ammonium bromide. Sections obtained from brains or spinal cords, which had been kept in formaldehyde solution for months or even years, when treated in such manner and then stained by any of the methods based on the original Cajal gold chloride-sublimate principle, yielded uniform and clear histologic pictures.

Another advantage in this new step is the opportunity to use sections thinner than those suggested by the authors of the original method. This insures greater clarity and uniformity of preparations, which are essential for detailed study and photomicrography.

DETAILS OF TECHNIC

1. Prepare frozen sections of formaldehyde-fixed material at a thickness of from 15 to 30 microns.

2. Wash quickly in several changes of distilled water.

3. Place in a 10 per cent solution of strong ammonia water for twenty-four hours at room temperature, or for shorter periods in an incubator.

4. Carry rapidly through two changes of distilled water.

5. Place in a 10 per cent solution of (pure, 41 per cent) hydrobromic acid, and let it remain there for from two to four hours.

6. Wash quickly in two changes of distilled water, to which a few drops of ammonia water is added.

7. Treat in accordance with the method selected. If it should be necessary to delay the final staining for some time, sections may be put into a 2 per cent aqueous solution of ammonium bromide to which a few drops of neutralized formaldehyde has been added. In this medium, the sections may remain for a longer period and may be kept in readiness to be stained at a later date.

ILLUSTRATION OF RESULTS OBTAINED

Gold Sublimate Method of Cajal.—Frozen sections of formaldehyde-fixed material are treated as already described, and are then stained according to Cajal's original method, as follows:

1. Wash the sections thoroughly but rapidly in several changes of distilled water.

2. Transfer to a glass dish containing about 30 cc. of the following freshly made mixture:

One per cent solution of gold chloride (Merck, brown), 10 cc.
Corrosive sublimate, 0.5 Gm.
Distilled water, 60 cc.

Allow sections to remain in this mixture for a number of hours, often as long as eighteen, until they acquire an intense purple color.

3. Place for about five minutes in a fixing bath, consisting of
Concentrated solution of sodium hyposulphite, 5 cc.
Distilled water, 70 cc.
Alcohol (95 per cent), 30 cc.
Concentrated solution of sodium bisulphite, 5 cc.

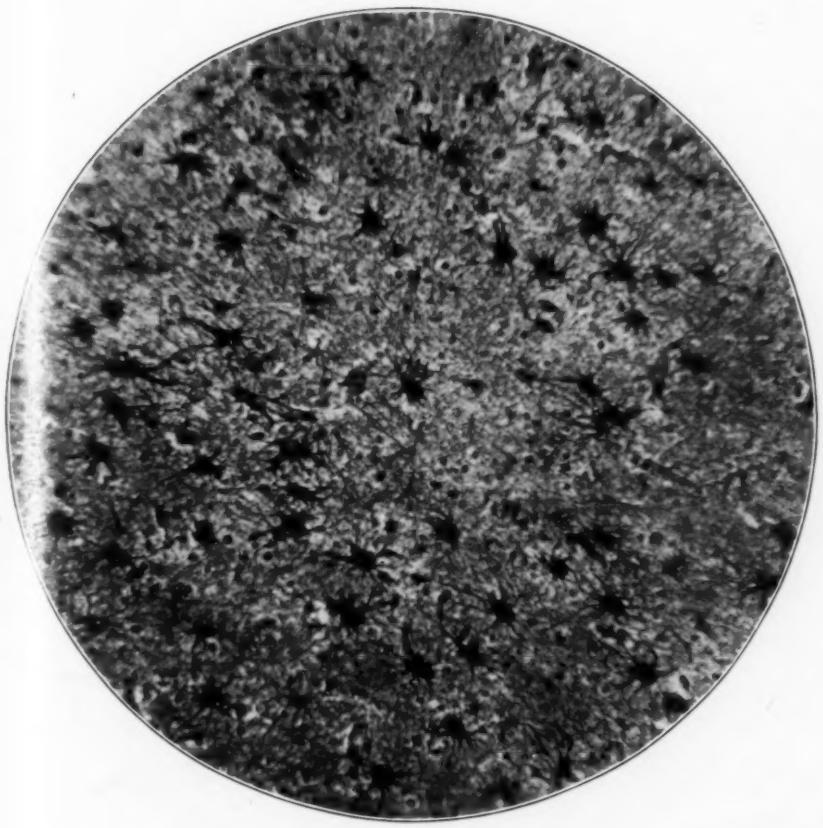

Fig. 1.—An average field showing both protoplasmic and fibrous astrocytes in an area of gliosis surrounding a hemorrhagic cavity. Modification of Cajal's gold chloride and sublimate method. × 160.

4. Transfer to 50 per cent alcohol, float on the slide, dehydrate in graded alcohol, clear with origanum oil, the excess of which is washed off by xylene, and mount in balsam.

This method brings into view an abundance of fibrous and protoplasmic neuroglia, and gives a good general picture of glial activity (fig. 1). It lends itself also to more detailed studies, when, for instance, the cellular origin of scar formation in the central nervous system is in doubt (fig. 2).

Del Rio Hortega's Carbonate of Silver Method (First Process).

1. Wash the frozen sections treated by the "new step" rapidly in several changes of distilled water, to which a few drops of ammonia water is added.

2. Place in 10 cc. of ammoniac silver carbonate solution. This is prepared as follows: To 50 cc. of 10 per cent silver nitrate solution, add 50 cc. of saturated lithium carbonate solution, precipitating the silver in the form of silver carbonate. Decant the fluid and wash the precipitate with a large amount (from 200 to 300 cc.) of distilled water. Dissolve the precipitate in about 50 cc. of diluted

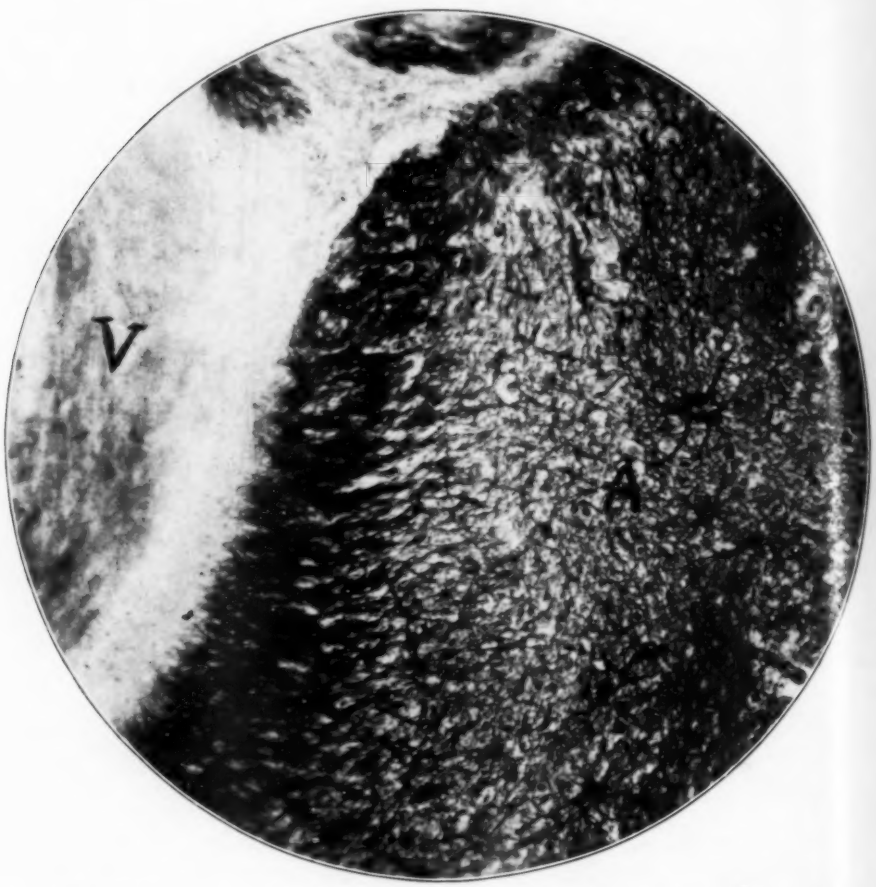

Fig. 2.—Dense glial fibrosis about a thrombosed blood vessel (*V*). A large fibrous glia cell (*A*) is seen contributing its processes to the felt-work around the vessel With a hand lens other smaller astrocytes are visible. Modification of Cajal's gold chloride-sublimate method. × 210.

ammonia. This solution is further diluted with distilled water, which brings the volume up to 250 cc. and is kept as a stock solution in dark brown bottles.

3. Keep the sections in the ammoniac silver carbonate solution in the incubator at a temperature not higher than 55 C., or keep warm over a flame until they become grayish-yellow.

4. While they are still warm wash quickly in distilled water and transfer into 20 per cent neutralized solution of formaldehyde. Allow the sections to remain in this solution for from two to three minutes, when they turn black.

5. Tone in gold chloride solution; fix in the hyposulphite bath; dehydrate; clear in carbol-xylene-creosote mixture on the slide (carbolic acid, 5 cc., xylene, 45 cc. and cresote, 50 cc.; wash in xylene and mount in balsam.

By this method, good pictures of fibrous and protoplasmic astrocytes are obtained (figs. 3, 4, 5 *A* and 6).

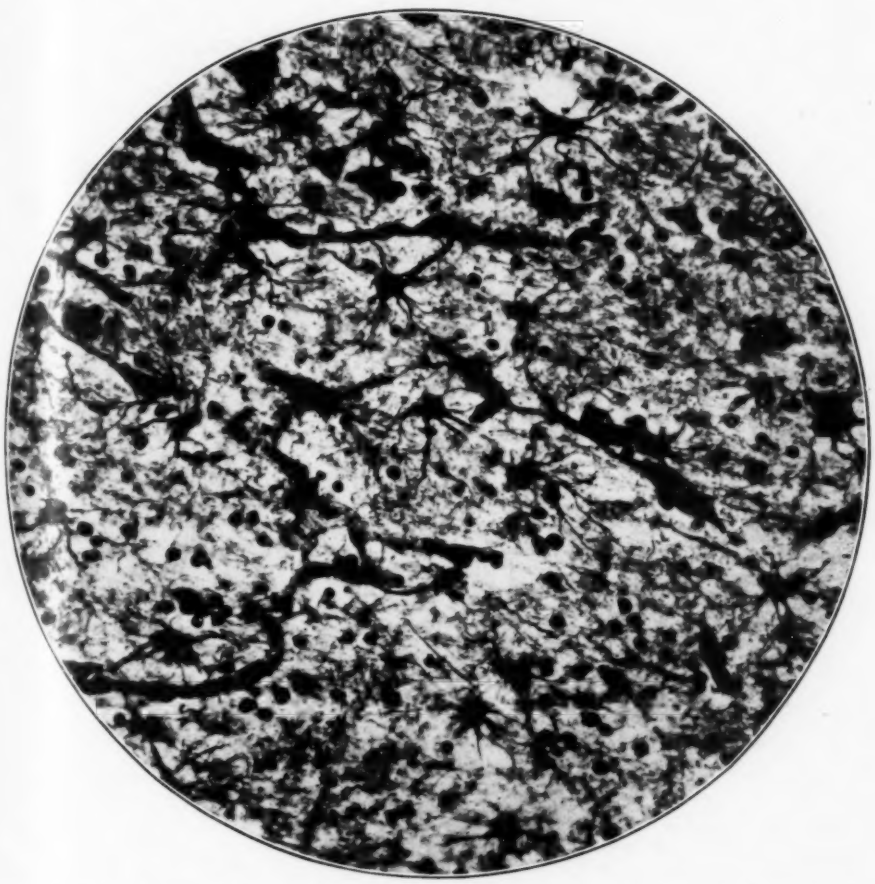

Fig. 3.—Gliosis in the vicinity of a hemorrhagic cavity, with numerous swollen fibrous astrocytes, whose vascular processes are well shown. Modification of Hortega's silver carbonate method, process 1. × 350.

Del Rio Hortega's Silver Carbonate Method (Third Process).

1. Place sections prepared by the newly described step for a few minutes in the following formaldehyde-ammonium bromide mixture:

Ammonium bromide, 2 cc.
Forty per cent solution of formaldehyde, 15 cc.
Distilled water, 100 cc.

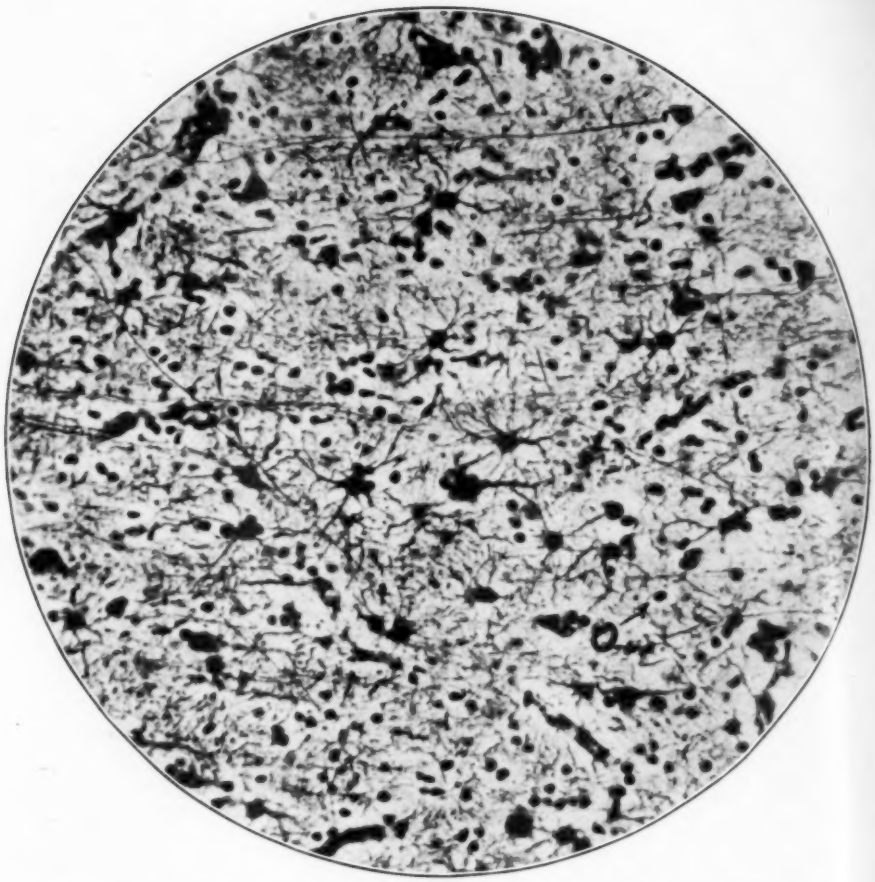

Fig. 4.—Gliosis in syphilitic meningovascular disease with many fibrous astrocytes dominating the field. There are also some oligodendroglia (*o*) cells, but they are poorly brought out. Modification of Hortega's silver carbonate method, process 1. × 230.

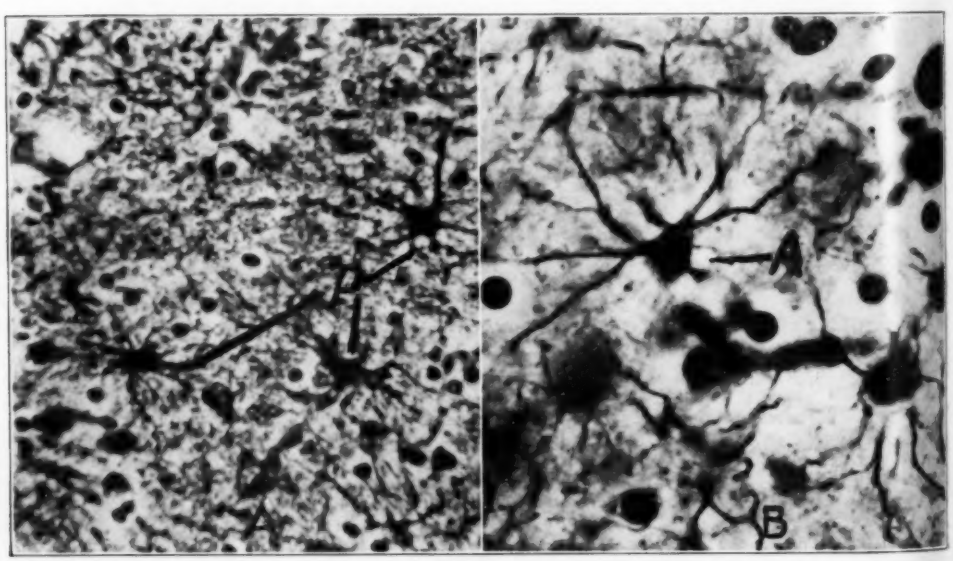

Fig. 5.—*A*, protoplasmic astrocytes (*P*) in an area of softening. Modification of Hortega's silver carbonate method, process 1. × 315. *B*, fibrous astrocyte (*A*). Modification of Hortega's silver carbonate method, process 3. × 725.

2. Wash rapidly in distilled water[3] and place in an ammoniac silver carbonate mixture, which is prepared in the following way: To 10 cc. of 10 per cent silver nitrate solution add 30 cc. of 5 per cent sodium carbonate solution. To the precipitate add gradually and carefully, drop by drop, strong ammonia until the precipitate is dissolved (avoid excess of ammonia). Add distilled water to bring the volume to 150 cc. Keep in dark bottles.

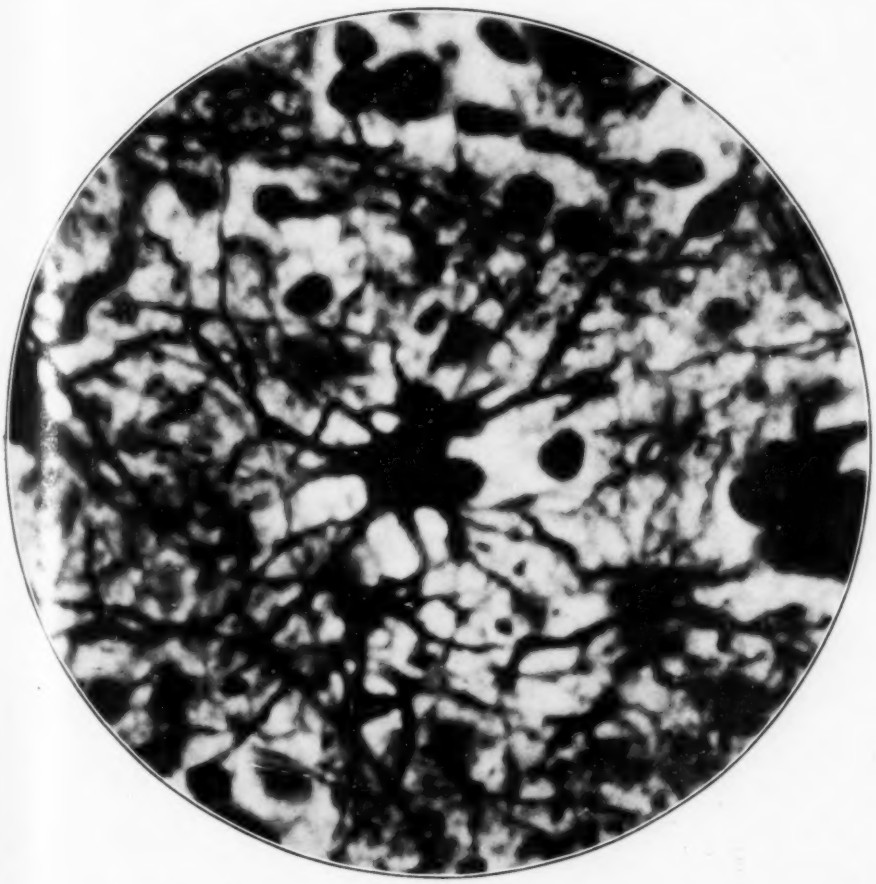

Fig. 6.—A giant fibrous astrocyte in an area of softening. Modification of Hortega's silver carbonate method, process 1. × 900.

3. Allow the sections to remain in this mixture for from twenty to thirty minutes at room temperature—or gently warm them over a flame. Reduce with 1 per cent neutralized 40 per cent solution of formaldehyde, allowing the sections

3. At the suggestion of Penfield, sections were allowed to remain in a 1 per cent solution of sodium carbonate for about five minutes before they were carried into the silver carbonate bath. This prevented the appearance of precipitates, and apparently helped impregnation.

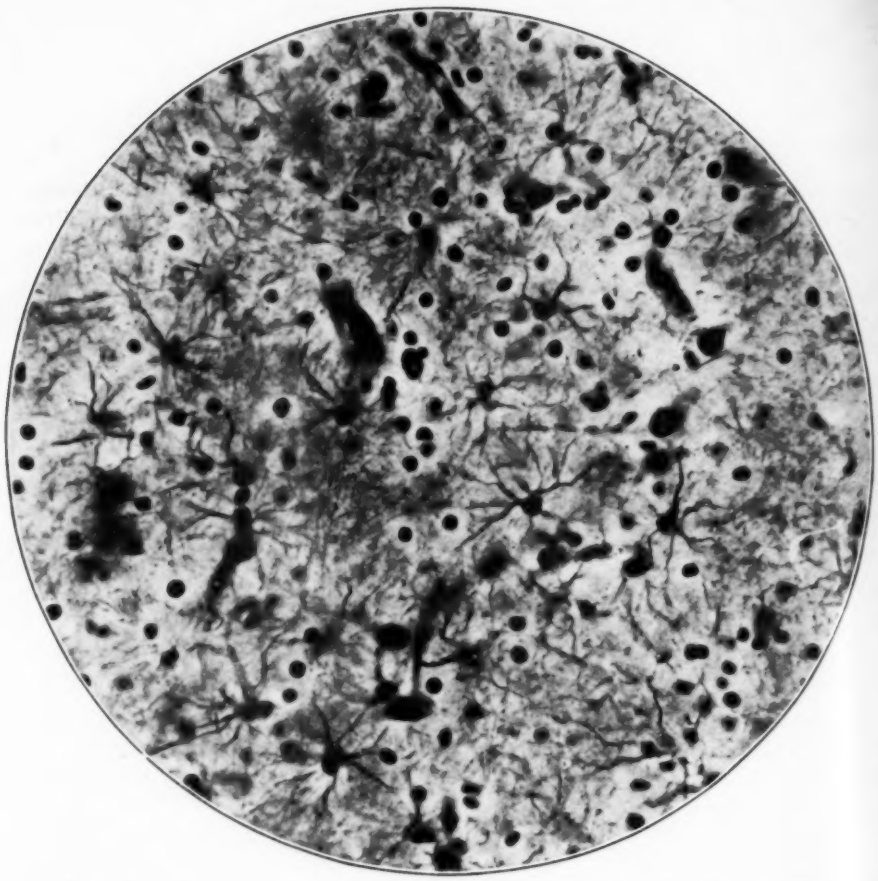

Fig. 7.—Fibrous astrocytes. Modification of Hortega's silver carbonate method, process 3. × 350.

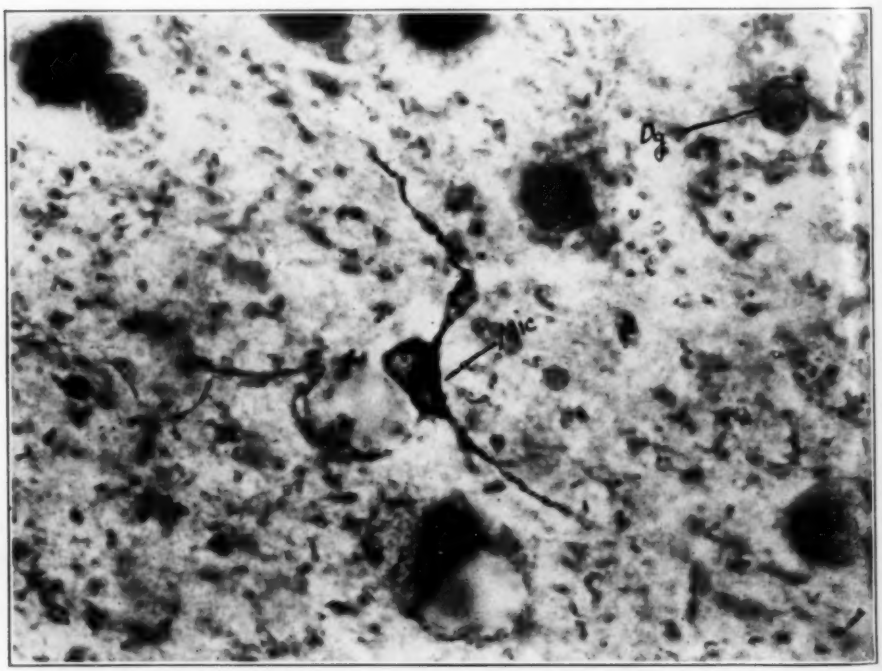

Fig. 8.—Microglia (*Mic*) and oligodendroglia (*Dg*). Modification of Hortega's silver carbonate method, process 3. Prepared and photographed by Dr. Penfield.

to remain in the solution until they become grayish-yellow. Tone in gold chloride solution, dehydrate, clear in formaldehyde-creosote-xylene mixture, and mount in balsam.

This method is designed by Hortega for the demonstration of microglia. In my experience, it yielded satisfactory pictures of protoplasmic and fibrous glia (fig. 7).

In the hands of Penfield,[4] who was kind enough to lend me a section and photomicrograph (fig. 8), microglia was brought out by my modification in material that was too old to be stained by the usual Hortega method.

CONCLUSIONS

The usefulness of the suggested new step can be seen in the uniformity and clarity of histologic preparations and in their adaptability to photomicrography, as well as in its availabilty for old material.

4. Penfield, W.: Brain **47**:430 (Dec.) 1924.

News and Comment

FORMATION OF COMMISSION FOR STUDY OF EPIDEMIC ENCEPHALITIS

A matter of more than usual interest has arisen in neuropsychiatry. It involves a comprehensive research in the epidemiology and treatment of encephalitis. This has been made possible by a generous patron of medical science, Mr. William J. Matheson, chemist and financier. He has given the income of a large fund set aside for this purpose.

A commission has been appointed to supervise this work. The committee acts under the provisions of the foundation and consists of:

Dr. William Darrach, chairman
D. Frederick Tilney
Mr. Willis D. Wood
Dr. William H. Park
Dr. Haven Emerson
D. Frederick P. Gay
Dr. Hubert S. Howe, secretary.

The committee has been able to obtain the services of Dr. Josephine Neal, who will direct the investigation.

The committee requests, through this journal, the active participation of all physicians, and particularly of those who have had experience in the management and treatment of patients with epidemic encephalitis. Any information or suggestion in the field of epidemiology, diagnosis and therapeutics will be thankfully received by the committee through Dr. Neal, at the Academy of Medicine, 2 East 103rd Street, New York City.

ANNOUNCEMENT OF AN INTENSIVE POSTGRADUATE COURSE IN NEUROLOGY AND PSYCHIATRY AT VIENNA, WINTER, 1928

A special systematic course for postgraduate study of neurology and psychiatry will be given entirely in English between Jan. 2 and Feb. 28, 1928, at the Neuropsychiatric Clinic of Prof. Wagner von Jauregg and at the Neurological Institute of Professor Marburg, Vienna University, Austria. The whole field of neurology and psychiatry and related branches (otology, ophthalmology, endocrinology, roentgenology, brain surgery) will be covered. Applications should be made to Docent Dr. E. Spiegel, Falkestr. 3, Vienna, I, Austria.

Abstracts from Current Literature

THE EFFECT OF ROENTGEN RAYS ON THE DEVELOPMENT OF THE BRAIN IN ANIMALS. (PRELIMINARY COMMUNICATION). RUDOLF DEMEL, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **28**:13 (May) 1926.

After exposing the brains of fully developed animals to roentgen rays, Brunner found that only immature (histologically) parenchymatous elements, such as the superficial cerebellar nuclei and proliferating glia, were affected by the rays, whereas elements like mature glia cells, ganglion cells and medullated nerve fibers, were unaffected. His histologic preparations showed only slight changes in ectodermal cerebral tissue but definite changes in the vessels and connective tissue; at the same time, there could also be seen considerable cerebral edema and moderate internal hydrocephalus. Brunner's animals also developed, after irradiation, epileptiform seizures and disturbances in growth with free hemorrhages in the brain substance and in the meninges as well as localized perivascular round cell infiltrations.

Following Brunner's work, Demel subjected thirteen young dogs from three different litters to roentgen rays; at the first irradiation the dogs of the first two litters were 4 days old, and those of the third litter 2 weeks old. The animals were irradiated in four sittings at intervals of four days; at each exposure each animal was given two thirds of a skin unit dose through a 3 mm. aluminum filter. In the six dogs of the first two litters (first litter, two dogs; second litter, four dogs) the entire skull was irradiated, the face and the rest of the body being carefully protected from the rays; in the third litter (seven dogs), the irradiation was carried out as follows: In one dog only the rhinencephalon was exposed; in another, only the cerebellum; in a third, only the right half of the skull; in a fourth, the right motor area; in a fifth, the left motor area, and in a sixth, both motor areas; one animal from each litter was used as a control.

The animals were anesthetized with ether and morphine before irradiation to insure their remaining quiet throughout the exposure, which lasted only seven minutes. Care was also taken that only the parts it had been decided to irradiate were actually subjected to the rays. The two dogs of the first litter survived the experiment longest (five months); the four dogs of the second litter survived two months, two months, sixty-five days and seventy days, respectively; those of the third litter survived nine, twenty-one, twenty-three, thirty-three and forty-one days, respectively; one dog of the last litter was still alive at the time of publication.

Study of the duration of life following irradiation brought out the fact that it depends apparently on the physiologic importance as well as on the size of the irradiated area, so that in the third litter the animal in which an entire hemisphere was irradiated survived nine days; the one in which both motor areas were exposed survived twenty-one days; the one whose right motor area was exposed survived thirty-three days; the one whose left motor area was exposed survived thirty-six days; the one whose rhinencephalon was exposed survived forty-one days, whereas the animal whose cerebellum was irradiated was alive and in excellent condition at the time of writing. Externally, the irradiated animals showed changes that were similar in nature but whose intensity depended greatly on the duration of life following the irradia-

tion. (1) The irradiated dogs showed marked disturbances in growth involving the entire skeletal system. Roentgenologically, the bones showed no structural deviations from the normal except that they were more slender and shorter; this change was evident in the fourth week after the last exposure. (2) After the first exposure, all animals began to show evidences of gradual emaciation which increased rapidly so that necropsy showed a total disappearance of the subcutaneous and peritoneal fat; the serosa of the intra-abdominal organs was lusterless and dry. (3) At the end of the seventh or during the eighth week, the irradiated animals developed a disturbance in gait, which was ataxic and on a broad base; there was also a marked diminution in motor power; as the condition advanced, the dogs could not stand still, and there was a tendency for the body to veer to one side; later, the animals could not even sit straight but kept falling to one side. These disturbances are probably due to organic changes in the brain and are in conformity with the observations made by Pfandler and Zappert, who found spasms, pareses, general rigidity, tremor, clumsiness and crossed legs with spastic gait in congenital hydrocephalus in human beings. Ophthalmoscopic examination of the animals in the first and second litters showed the following changes in the sixth week after exposure: neuritic atrophy similar to that observed in man; no ophthalmoscopic changes in the animals in the third litter, the duration of life being probably too short to produce changes in the disks.

Macroscopically, the irradiated brains showed a general diminution in size involving cerebrum, cerebellum and pons. The weight of these brains was considerably less than that of the control animals. The external configuration of the gyri was undisturbed, although the sulci appeared shallower and more concentrically arranged; the occipital lobe in the animal from the first litter that survived five months showed pachygyria, the convolutional picture being fetal in type. The hippocampal region was relatively well preserved. This would seem to indicate that the phylogenetically older parts of the brain are more resistant to roentgen rays than phylogenetically younger parts.

Microscopically, the longest surviving animal showed the following changes: Section 1, through the medulla, showed a marked hemiatrophy on the right side; the pyramids and restiform body were much smaller on that side; the right side of the area under the fourth ventricle was poorer in fibers, especially in the region of the vagus nucleus; otherwise, the histologic picture was normal. Section 2, through the medulla and the beginning of the pons showed hemiatrophy on the right side with a general diminution in the size of the cerebellum and definite malformation of that structure as well as of the vermis on that side. Section 3, through the corpora quadrigemina showed definite diminution in the size of the right side, especially at the beginning of the right peduncle; the periaqueductal area also appeared smaller; except for a slight diminution of fibers in the region of the aqueduct and under the fourth ventricle, the histologic picture was normal. Section 4, through the corpora geniculata showed a definite hemiatrophy on the entire right side, especially of the cerebral peduncle and of the mesial geniculate body. Section 5, through the optic thalamus showed a striking reduction in the size of the right thalamus and cortex and a moderate hydrocephalus on that side. Section 6, at the level of the basal ganglions showed marked atrophy of the right hemisphere; the basal ganglions themselves were normal in size. Section 7, through both frontal lobes showed atrophy of the right side and a peculiar fiber picture of the cortex characterized by the presence of myelinated and nonmyelinated areas, the latter being bilateral. The hemiatrophy of the entire central nervous

system, limited as it was to the right side, was apparently in harmony with the fact that the irradiation was limited to that side of the brain.

DETAILED HISTOLOGIC STUDY OF THE CORTEX

The same brain was examined. (1) Section through the frontal cortex—gyrus sigmoideus showed all layers of the cortex to be distinct and normally arranged on the more atrophied right side; in some areas, the cells in some of the layers had disappeared; this was especially noted in the upper layers; the molecular zone actually appeared wider; in other areas, the cell destruction was much more extensive and more diffuse. There was no evidence of any inflammatory process nor of any vascular or glial reaction. The meninges, however, showed a moderate production of cells, especially near the vessels. The less atrophied side showed the cortex to be of normal structure, but there was considerable diminution of the cells, especially in the upper layers; there was also present a marked degeneration of the ganglion cells (vacuolization). (2) Section through the parieto-occipital region showed all layers of the cortex on the more atrophied side to be fairly well intact, except for a few areas in which the superficial layers appeared somewhat shrunken; the molecular zone appeared almost twice its normal size; this was at the expense of the superficial layer; the cells in this region were also severely affected (swelling, shrinkage and destruction). The glia also showed a moderate reaction. The less atrophied side at this level showed a picture similar to that in the frontal cortex on the same side.

The retina in the irradiated dog appeared normal only in some areas; its periphery showed complete degeneration with roset formation; in some areas only a few glial fibers remained intact. There were no inflammatory changes and no atrophies of the pigment epithelium.

This piece of research would seem to indicate that the fetal or early infantile nervous system reacts to external noxious agents differently from the adult nervous system. D'Abundo has also shown that infants' brains develop hydrocephalus after the slightest lesions in the nervous system, regardless of where these may be located. The nearer the lesions are to the ventricle, the more marked is the hydrocephalus. Up to the present time, cerebral hemiatrophy was regarded as a congenital malformation due to an injury to the germ plasm. Demel's investigation shows that hemiatrophy may also be due to exogenous factors. Further studies on the effect of roentgen rays on the fully developed brain are now in progress, but these will be the subject of a future communication.

KESCHNER, New York.

REFLEXES OF SPINAL AUTOMATISM. AXEL OWRE, Acta psychiat. et neurol. **1**: 260, 1926.

The reflexes of spinal automatism, which occur when the spinal centers are liberated from cerebral control, manifest themselves as synergic flexion and extension movements of the extremity and are elicited best by stimuli that provoke a sensation of pain in the normal organism. The most constant of these reflexes is the flexion reflex, which, under definite conditions, may be accompanied by the crossed extension reflex. In the normal person, involuntary withdrawal of the leg in response to painful stimuli applied to the sole of the foot may resemble a flexion reflex, except that the big toe will be plantar flexed instead of dorsiflexed. The true flexion reflex shows dorsal flexion of the foot at the ankle following stimuli applied elsewhere than to the sole of the foot.

The receptive field for these reflexes includes all types of tissue in the extremity, and the author considers that the best manner of stimulation is pinching the skin and pricking or stroking with a pin. The intensity of the stimulus necessary to produce a reflex movement depends on the reflex irritability of the patient. In compressional processes and multiple sclerosis this irritability is excessive, which Babinski attributes to intramedullary changes. Toxic processes, such as bed-sores, pyelocystitis, etc., decrease it. The irritability is abolished for a time by traumatic or other suddenly occurring cord lesions. In general it may be said that the stronger the stimulus the greater will be the reflex movement and that weak stimuli may be effective by summation.

The optimum site for the application of the stimulus is the lateral side of the sole of the foot; next the dorsal aspect of the foot and the lower third of the leg, the inner side of the thigh and the perineum. In the cases in which the reflexogenous zone extended to the abdomen it was easier to elicit the reflex from the medial than from the lateral side of the leg. The extent of the reflex zone corresponds to the reflex irritability of the patient. The approach of a fatal termination is marked by the gradual decrease in extent of the zone and its ultimate confinement to the sole of the foot. This decrease may begin only a few hours before death.

Dorsiflexion of the big toe and ankle is maintained as an integral part of the flexion reflex until the stimulus is applied to the thigh. Stimulation here gives flexion at the knee and hip as its most prominent feature—the local sign of Sherrington. This is the main form of the reaction following stimulation of the abdominal area also. At the limits of the reflexogenous zone the direction of the movement of the ankle and big toe changes, flexion of the hip and knee being accompanied by plantar flexion of the toe and ankle. When the stimulus is applied below the knee, dorsiflexion of the big toe is an integral part of the reflex; when it is applied above the knee, the response may be plantar flexion. The author does not agree with Walshe and Riddoch that the reflexogenous zone of the crossed extension reflex is identical with that of the flexion reflex. He found that the zone from which the former was elicited most frequently was the distal part of the contralateral extremity.

In the normal person, the pyramidal and extrapyramidal systems exercise a tonus-subduing influence and the cerebellum, labyrinth and the proprioceptive impulses from the periphery exert a tonus-increasing influence. The tonus of the erect posture is composed of a series of spinal and prespinal reflex arcs the centers for the latter being situated in the midbrain, and, in man, seeming to have a superior functional control, so that when the spinal cord is severed the reflex tonus disappears from the extremities. In complete transverse lesions the extensor tonus should disappear and only flexor reflexes be obtained; after the period of shock, the tonus and reflex irritability returns to the muscles of the flexor groups first. Only later and to a less degree does it return to the extensors. Extensor tonus is not attached so exclusively, therefore, to the extrapyramidal system as is postural tone, neither is it of cerebral origin.

The author inclines to the theory that the solution of the problem of tonus must be sought in the sympathetic innervation of the muscles. He attributes the great increase in tonus in incomplete transverse lesions of the cord to spasm of the sarcoplasmic element of striated muscle, while the aplasticity or hypoplasticity of complete transverse lesions is traceable to rupture between the upper and lower centers of the sympathetic nervous system. The contractile element of the muscles will be present in both instances. On the basis

of this theory, tendon phenomena can be divided into two phases: a rapid contraction due to action of the myoplasm and a tonic maintenance of the contraction by the sarcoplasm. This explains the prolongation of the patellar reflex in incomplete transverse lesions and the sudden aplastic jerk in complete transverse lesions. There is no answer, as yet, to the question of the cause of the increased tonus in the latter condition.

Incomplete transverse lesions may result either in paraplegia in extension or in paraplegia in flexion. In the latter instance, active movements are decreased more than in the former, the tendon reflexes are increased and show an aplastic relaxation rather than the prolonged relaxation characteristic of the former, and the reflexogenous zone for the reflexes of spinal automatism is extended greatly—reaching to the abdomen in many cases (in paraplegia in extension it seldom extends beyond the knee). The author considers the flexion condition to be more common in compressional processes. A case commencing as paraplegia in extension in time may become one of paraplegia in flexion, this change indicating a progressive decline in the patient's condition. The flexor position may terminate in a stable flexor contracture.

Following a complete transverse lesion, a period of spinal shock supervenes, of about two weeks' duration; then the reflexes begin to return gradually. During this stage of return of the reflexes the muscles appear atonic, but in reality they have a certain amount of tone. The relaxation stage of the patellar reflex is sharp and aplastic, and the reflexogenous zone for the reflexes of spinal automatism is extended. The reflexes resemble those of paraplegia in flexion, but are still more rapid than in that condition.

Clinically, it is important to recognize certain of these reflexes. It is necessary to realize that spontaneous activation of the flexion reflex is the forerunner of a flexor contracture, which should be prevented for the comfort of the patient. Its presence indicates a decline in the patient's condition. If plantar flexion of the big toe follows the stage of shock the prognosis is bad, as it indicates that the patient has a complete transverse lesion. The prognosis is bad also if the extremities become flaccid two or three days after the lesion, while maintenance of tonus and a quick return of the reflexes indicate that improvement may be expected.

The presence of these reflexes may indicate the extent of the lesion. If the upper level may be determined by the sensory loss, the highest level from which these reflexes may be elicited may be considered the lower level of the lesion, as Babinski and Jarkowski pointed out. This is not always the case.

The author examined the majority of his patients in a warm bath as well as in bed and concludes that the results are the same by the two methods, though in the latter, care must be exercised to prevent the heels rubbing on the bed and the cooling of the limbs.

PEARSON, Philadelphia.

THE MALARIAL TREATMENT OF TABES DORSALIS. HANS HOFF and OTTO KAUDERS, Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:306 (Aug.) 1926.

While the treatment of tabes before the use of malaria was more or less inefficient, progress had been made by the use of certain drugs. It took a hopeful rise after the introduction of mercury by Erb, and while neoarsphenamine has been discredited by Oppenheim and Strümpell, it too has had its place in the specific treatment of tabes dorsalis. The extremists against the use of arsphenamine in tabes are represented by Dreyfus, Leredde and Gennerich. The French, Lhermitte and Levy, Sicard and Mestrezat, favor the use of subdural

and epidural injections. The treatment of tabes, however, made a definite advance through the use of protein therapy as advocated by Wagner-Jauregg who used tuberculin.

There are few reports on the treatment of tabes by malaria. Weygandt, Nonne, Herman, Leese and Peter, McGrath and others have reported on this treatment. Bering has reported favorable results in his series. Hoff and Kauders report fifty-six patients having tabes dorsalis who were treated with malaria; the longest duration was for four years and the shortest for from six months to one year. Cases were selected which showed only a slight tendency to progression, while cases of taboparalysis and of tabetic optic atrophy were excluded. Careful investigation of the general status of all the cases was made. An analysis of the treated patients was made first from the standpoint of the outstanding symptoms. The cases were divided into: ataxic forms, ambulatory and nonambulatory; lancinating pains; gastric crises.

Analysis of the cases from this standpoint shows that only 25 per cent were uninfluenced by malaria treatment, and the different forms were influenced differently. The least favorable reaction was in the severe ataxic group. In this group (twenty-two cases) 62 per cent were influenced, while the other 38 per cent were not markedly affected. Of the slightly ataxic forms (eight cases), only 25 per cent were uninfluenced, 25 per cent were markedly improved, and 50 per cent slightly improved. The most favorably affected groups were those with lancinating pains and gastric crises. Only 11 per cent and 12 per cent of these cases, respectively, were not influenced by treatment. About 63 per cent of cases in which gastric crises occurred showed marked improvement or cure of these attacks. In 39 per cent of the cases of lancinating pains, the pains were entirely cleared up, while 50 per cent showed a marked improvement.

Analysis from another angle shows that the fresh cases of tabes dorsalis react more favorably to malarial treatment than do the chronic progressive forms. Only 10 per cent of the fresh cases (ten cases) were uninfluenced by treatment, while 28 per cent of the chronic progressive forms (forty-six cases) were uninfluenced. The percentage of complete and partial "cures" is also 9 per cent higher among the fresh than among the chronic forms.

The view that cases of tabes showing markedly positive serologic symptoms give the best chances in treatment is not substantiated by Hoff and Kauders. They obtained good results in cases showing weakly positive to negative serologic symptoms. Those with strong serologic reactions showed no greater tendency to remission or cure than did those with weakly positive or negative reactions. In three cases the blood Wassermann reaction became negative after malarial treatment, either immediately or shortly after treatment. It is the rule, moreover, in strongly positive fluids for the cell count to be greatly decreased or to return to normal. The globulin and albumin content of the fluid is markedly decreased. The effect of malarial treatment on the Wassermann reaction of the spinal fluid was practically negligible in Hoff and Kauders' cases. In two cases, it fell from strongly positive to weakly positive. Follow-up after the end of malarial treatment showed that 52 per cent of the patients who had improved had made further progress after treatment had been stopped. In 36 per cent the condition was the same as just after treatment, while in only 12 per cent was there a decline.

Bering reported twenty-three patients treated with malaria. Of these, four patients, or 14 per cent, were not influenced by treatment; nineteen patients, or 86 per cent, were much improved. Bering observed in several cases a return of the patellar reflexes, but Hoff and Kauders could not substantiate this in their series.

The malarial treatment of tabes, just as in general paralysis, has its effect on the general bodily condition of the patients. They feel subjectively better and stronger and show an increased vitality. Most of them gain weight. One severely ataxic patient showed an increase of 13 Kg. in weight and still maintained this increase at the end of two years. Many of the patients are capable of returning to work, and this is especially true of those having gastric crises and lancinating pains.

With respect to isolated symptoms, the authors found no change in the pupils as the result of treatment. Two cases of paralytic squint of several years' standing did not show any change, while two cases with fresh oculomotor palsies were cleared up. In one case an abducens palsy developed during treatment, but this disappeared after treatment was stopped. Two patients having tabes with typical attacks of migraine were cured of the attacks by malaria. The reflexes in the lower extremities were uninfluenced by the treatment. The effect of malarial treatment on paresthesias and on the disturbances of superficial and deep sensibility is slight. In a few cases, the improvement in ataxia was accompanied by some improvement in sense of position. In most cases deep sensibility was uninfluenced. Sphincteric disturbances were markedly improved. In accompaniment with increase in tone elsewhere in the body, there was a corresponding increase in the bladder and sphincter tone. In the majority of cases disturbances of the bladder were improved. The improvement in the slight degrees of ataxia has been noted, while severe ataxia was practically unaffected.

ALPERS, Philadelphia.

ATTEMPTS TO CURE OR TO INFLUENCE PSYCHIC DISEASES BY MEANS OF SHOCK REACTION. JULIUS SCHUSTER, Deutsche Ztschr. f. Nervenh. **95**:172 (Dec.) 1926.

To Wagner von Jauregg we owe a positive means of influencing favorably at times the course of certain types of syphilis, though long ago physicians observed that certain types of patients, especially those with psychic disturbances, were favorably influenced and at times entirely cured following an intercurrent infectious disease. Schuster, following Jauregg's idea, attempted to influence psychic disturbances by the injection of foreign protein in sufficient doses to produce a shock reaction. In a previous article (*Arch. f. Psychiat.*, vol. 77, 1926, p. 314) Schuster reported the cases of several patients successfully treated by anaphylactic shock methods. In this article Schuster reports the cases of eleven patients treated by the same method with satisfactory results.

A young man, who, soon after the death of his father, developed visual and auditory hallucinations, and became excitable and disoriented with ideas of persecution, received three milk injections, 10 cc. each, ten days apart. He developed a fever of 104 F. which kept up with variations for two weeks. At the end of two weeks he began to improve and in a short time recovered normal health.

A woman, aged 23, who had had many stormy love adventures since puberty, and had been depressed after the first disappointment at the age of 13, had suffered for the last six years from visual and auditory hallucinations, delirium and imperative ideas; she thought her body was not clean; the uncleanliness spread to any one touching her and she washed herself daily for six hours; speech also became paraphasic. Later she became careless and demented. She was given four injections of milk, 10 cc. each, ten days apart. The fourth injection caused a marked anaphylactic reaction. The temperature rose to

between 103 and 104 F., and there was considerable sweating and salivation. She lost 8 Kg. in weight in forty days. With the onset of the reaction the hallucinations ceased, the mind gradually cleared and the patient left the hospital entirely recovered.

A girl, aged 22, developed an acute manic attack after meeting a former lover who had married another woman. She became restless, disoriented, violent, did not speak, refused food and lost considerable weight. After several months, when no improvement was noticed, she was given four injections of milk. The first was given intramuscularly, the second and third subcutanously and the fourth (1.5 cc.) intravenously. After the last injection the temperature stayed up for about two weeks. The psychic change was crisis-like. In one week the patient became perfectly oriented and left the hospital in a short time.

A man, aged 31, developed a state of depression for the third time. After several injections of milk, he recovered completely. In the last injection 1 cc. of 5 per cent peptone was added to the milk.

A woman, aged 31, disoriented, hallucinating, excitable, and with ideas of persecution, gradually recovered under the shock method treatment.

A man, aged 27, suffered from headaches for two years, tired easily, lost interest in things, potency became diminished, movements became slow and he kept to himself; later he became restless, quarrelsome, irritable and developed auditory hallucinations. This patient recovered entirely after injections of foreign protein.

A woman, aged 34, suffered from severe melancholia. Six years before, she had had a similar depression with suicidal tendencies which had lasted one and one half years. She was given injections of milk, with excellent results. Another woman, aged 21, with marked manic symptoms yielded promptly to the treatment. A woman, aged 37, with severe melancholia, a woman, aged 21, with delusional insanity, and a woman, aged 51, with ideas of persecution promptly recovered under the anaphylactic shock treatment.

The eleven cases detailed in this article, together with five reported previously by Schuster, in which the patients were successfully treated by anaphylactic shock, are more than suggestive. The fatal case reported by Schuster should warn against the indiscriminate use of foreign protein, especially in doses capable of producing anaphylactic shock reaction. It cannot be said that sixteen successful cases is a sufficient number to justify an opinion.

There are many questions yet to be answered. What is the etiology and pathology of the mental disorders treated? If anaphylactic treatment is successful, must we postulate the presence of an intoxication or an infection? Schuster comes to that conclusion. According to him, many psychic disturbances may depend on either one of two components. One is a toxin which affects the capillary walls and the centers that regulate the brain capillaries, and damages the ganglion cells and glia in the cortex, basal ganglia and vegetative centers. It causes a change in the capillary circulation of the entire organism. Another component may be one which for any other reason causes change in the capillary circulation in the brain and other organs of the body. Schuster must have in mind, when he speaks of the non-toxic type, cases such as the girl who developes acute mania immediately after meeting a former lover. Such cases cannot be due to toxic conditions as ordinarily understood. The question then arises as to the manner in which a foreign protein operates in such a case. Does a sudden mental shock, strong enough to upset the psyche, produce a change in the glands and the vegetative nervous system that gives rise to a

secondary acute toxic state, or without primarily affecting the vegetative centers cause an acute toxic state acting directly on the nerve centers? It would be interesting to know how long Schuster's patients remained cured.

BERNIS, Rochester, N. Y.

THE ANATOMY AND PHYSIOLOGY OF THE LABYRINTH OF THE EAR AND OF THE EIGHTH NERVE. Part 1. L. R. LORENTE DE NO, Trav. du lab. de rech. biol. de l'université, Madrid **23**:259, 1925.

The author has studied the relations between the ocular movements, on the one hand, and the tonic neck and labyrinthine reflexes on the other. The method of extirpating one eye and recording the movements of the other eye was found inadequate, and a special technic was evolved of tracing the contractions of each individual eye muscle. The operative procedure and apparatus employed are described in detail. Eleven rabbits were used and a wealth of valuable data was obtained. As a preliminary, the theories of Magnus and de Kleijn, Quix and Breuer are discussed in broad outline. Their conclusions, largely based on Sherrington's law of "reciprocal innervation," are rejected as untenable, because too schematic. Magnus and de Kleijn hold that the ocular muscles are arranged in three pairs of antagonists, corresponding to the three rotation axes of the body: the rectus internus and externus to the dorsoventral axis; the rectus superior and inferior to the longitudinal axis, and the obliquus superior and inferior to the sagittal axis. However, as the author points out with convincing evidence, all six muscles of the eye must be regarded as a unit, acting as such in all movements.

Breuer had formulated the theory of two distinct labyrinthine organs: the cristae of the semicircular canals, on the one hand, and the maculae of the utricle and saccule on the other. Each of these labyrinthine organs is in correlation with a definite muscle or muscle group of the eye. But the author polemizes against a too dogmatic schematization and emphasizes particularly the central regulation (medullary and midbrain) of the involved reflex mechanism. Excitations proceeding from the labyrinth give rise to nervous impulses in the medulla whence they are transmitted to the motor centers of the eye and of the body. In an analogous manner the dynamic reflexes of the semicircular canals originate.

Though many facts are in favor of a separation of macular and semicircular function, proof is as yet missing. The assumption that the semicircular canals give rise to dynamic reflexes and the maculae to postural reflexes, is of doubtful origin. At present, we know only that certain tonic reflexes have their origin in the labyrinth, but whether in the maculae (otoliths) or in the semicircular canals is uncertain. Most likely they are produced by the excitation of both the canals and the maculae, and are integrated and modified by the medullary centers. The mechanism of integration is of enormous complexity and the organs involved in the integration process of confusing diversity. They are: the optic and vestibular centers, the muscle sensibility of the neck and eye, the motor nuclei of the eyes, and the cortex.

Each animal, even each individual muscle, has its specific character of contraction, dependent on the anatomic structure of the muscle. In addition, in young animals, the tonic movements are different from those in adult animals. The inference is that, at birth, there are no definite connections yet established between labyrinth and eye muscles, on the one hand, and the nervous centers on the other. The functional adjustment of labyrinth and eye muscles evolves during ontogenetic development.

It is impossible to do full justice to the thoroughness and completeness of the work incorporated in this study. The numerous curves and illustrations, together with the detailed and unusually painstaking recording of experimental data, must be read in the original. But the reviewer feels that the author engages in superabundant polemizing which is too often beside the mark. Paragraph after paragraph is devoted to a more energetic than fruitful criticism of Sherrington's law of "reciprocal innervation." Whole passages are directed against the assumption by experimental workers that the eye has a center of rotation independent of its individual muscles, against the conception and treatment of muscles as straight lines with points of origin and insertion. After all, such abstractions are the backbone of experimental thinking, and every experimenter is well aware that these abstractions, being without actual correlates in the world of reality, are used only as facilitating and simplifying symbolisms. As such, they yield results, in spite of their fictional nature. When the author, in further criticism, postulates that movements of muscles must be tested under "physiologic conditions," he simply ignores the nature of experimental investigation. Experimenting means precisely to create—consciously—unphysiologic conditions in order to isolate phenomena and functions which, in their state of physiologic integration, remain hidden from view and defy recognition.

Low, Chicago.

CLINICOPATHOLOGIC FEATURES OF VENTRALLY SITUATED TUMORS OF THE SPINAL CORD. FRIEDRICH KORNER, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **28**:51 (May) 1926.

Among sixty cases of ventrally situated tumors of the spinal cord collected from the literature, Korner could find only fifty-one cases with an anatomic diagnosis. Of these, nineteen were sarcomas, thirteen endotheliomas, six neuromas, four fibromas, one neurofibroma, five psammomas, one chondroma, one tubercle and one gumma. When these statistics are compared with those gathered by Anton, it would seem that the former figures for sarcoma are unusually high, and that endothelioma and neuroma are apparently the predominating type of tumor in this localization. It must also be emphasized that the tumors in most of the cases with this localization are more or less benign and offer excellent chances for surgical removal. From the point of view of size, these growths present no particular operative difficulties; the smallest tumor reported was the size of a hazelnut; in another case, the tumor was 1 cm. in diameter; ordinarily, tumors in this localization are the size of a pigeon's egg or a large walnut; their length usually exceeds their width; at all events, they are never as large as tumors situated on the dorsal or dorsolateral aspect of the cord. It would also seem that ventral tumors give signs earlier than dorsal tumors, i. e., before they attain considerable dimensions. Most of them extend only over one segment, some over two, and the largest of them have been found to extend over four segments. They are most commonly found in the lower cervical and upper dorsal regions. Of fifty-three cases reviewed by the author, there were twenty-one tumors in the cervical, thirty-one in the dorsal, and only one in the lumbar region. In the case reported in this paper, the tumor was in the lower dorsal and upper lumbar regions.

Pain is an early symptom; it may appear as early as in dorsally situated tumors; it may be severe and may temporarily disappear for a considerable period. The sensory disturbances may resemble those of tumors situated dorsally or laterally, or they may resemble those of intraspinal growths. There

may occur a total loss of sensation as in a tumor growing from the posterior aspect of the cord which destroys the sensory tracts by compressing them from without, or sensory disturbances may appear in the form of a band-like zone of slight hyperalgesia as in intraspinal lesions; if such a band-like zone is present, it is never isolated but is continuous below with a slight hypalgesia, which involves the rest of the body—a condition encountered in the later stages of intraspinal tumors.

Owing to the localization of these tumors in the thoracic cord, muscular atrophies play an insignificant rôle in the clinical picture, although they are occasionally encountered in tumors of the lower cervical or upper dorsal regions; only six patients in the series described had muscular atrophies affecting chiefly the thenar, hypothenar and interossei muscles. When atrophies are present, they are usually slight and rarely extend to the forearm or shoulder. The results of spinal puncture are the same in these growths as in those located on the dorsal aspect of the cord; the same is true of changes in the vertebral column.

After these preliminary remarks, Korner reports in detail a personally observed case that came to laminectomy during which a fibro-endothelioma was found and removed; the patient died three months later of decubitus; necropsy revealed at the original level, but on the posterior aspect of the cord, the beginning of a recurrence; histologic examination of the latter revealed the same anatomic structure as that of the tumor that had been removed, except that the tissue of the former was embryonic in nature. A similar case has also been reported by Eiselsberg and Marburg. These facts must be borne in mind when one gives a prognosis in these apparently benign tumors. The clinical picture of the case reported in this contribution pointed to a localization of the tumor on the dorsal aspect of the cord. The pain and the initial spasmodic twitchings with the total loss of sensation from the upper lumbar to the lowest sacral segments was so typical of a dorsally situated tumor that prior to laminectomy no other localization was suspected. It would seem then that the clinical course of a ventrally situated tumor may closely simulate that of a dorsally situated tumor. There are apparently no positive differential criteria that would enable one to diagnose a ventrally situated tumor with any reasonable degree of certainty.

KESCHNER, New York.

ROENTGEN-RAY THERAPY IN NEURALGIAS. AUGUST MÜLLER, München. med. Wchnschr. **73**:1915 (Nov.) 1926.

The treatment of true neuralgia is a thankless task. In the attack of acute pain, application of heat still promises the best results, but the recurrence of severe attacks drives the patient to the surgeon for permanent relief. Injection of sodium chloride solution, alcohol injection, resection of nerves, or, in trigeminal neuralgia, injection or resection of the gasserian ganglion are the methods of treatment employed at the present in the majority of cases. Gocht was the first to introduce roentgen-ray therapy in the treatment of neuralgia, in 1897. The best results are obtained in trigeminal neuralgia, but even here surgical intervention is resorted to before a final attempt has been made with the roentgen ray. The chief reason for this is the old fear of roentgen-ray burns, but with the present improved technic and knowledge as to exact dosage, etc., this fear is disappearing.

As Lentz pointed out in 1920, it is not a question of roentgen ray or operation; the treatment that is least dangerous should be tried first; certainly this

is the roentgen ray. Furthermore, if radiation is not of any benefit, an operation can still be performed. On the other hand, operation of any sort, even injection, so alters the nerve as to render future radiation useless. The author suggests that after pain has been relieved by hot applications roentgenotherapy should be employed; only when there are negative results with radiation should resort be had to surgery. The roentgen-ray therapy should be considered useless only when, after two treatments, there has been neither relief nor reaction in the sense that the pain is worse for several hours or at most a day.

There is still uncertainty as to the exact action of the rays on the nerve. In general it is known only that every living cell, according to its sensitiveness, is influenced by it. How to explain the action of the roentgen rays in trigeminal neuralgia is at present unclear. Those opposed to the hyperemia theory constantly refer to the fact that to produce hyperemia simpler means are available, for instance, diathermy. That is true, but with the diathermy apparatus a peripheral hyperemia is produced which does not touch the gasserian ganglion. By applying the roentgen ray to this region, however, a capillary hyperemia may be produced, which alleviates pain.

The author is inclined to believe that the hyperemia caused by the roentgen ray is responsible for the relief of pain in other conditions also, such as in dry pleurisy. Freund's theory is that the hyperemia causes a reduction of mechanical irritation of the nerves and carries off inflammatory products. Kottermaier says that the rays cause increased exudation in the finest lymph spaces which surround the nerves; these exudations cause compression and subsequent lessened possibility of entrance of the sensitive fibers. The author believes the theory of Freund to be the most logical since it tends to explain the reaction pains which regularly occur after radiation. These general reaction pains occur so regularly that the patient is apt to discontinue further treatment. On the basis of the author's experience with twenty-three patients, he has observed that the newer the case the more stormy the reaction—the pains of several hours up to one or two days' duration presenting the most favorable prognosis.

The technic is of the utmost importance. An instrument for deep therapy with which dosage may be accurately measured is essential if this valuable therapy is to have due credit.

In view of the splendid results obtained with the roentgen ray in trigeminal neuralgia, it is difficult to explain the lack of results in sciatica. Assuming that a pure neuralgia is being dealt with, equally good results should be obtained in the sciaticas, but this is not the case. Walter and Lax had no results with the therapy in sciatica, and in the author's seven cases of sciatica the results were inconstant. However, one patient, who had been confined to bed for a long time, was able to walk with a cane after roentgen-ray treatment.

In conclusion, the author states that roentgenotherapy should be tried in all cases of neuralgia, especially trigeminal, before surgery is resorted to.

MOERSCH, Rochester, Minn.

A STUDY OF THIRTY-FOUR CASES OF RAPIDLY DEVELOPING SYPHILITIC PARAPLEGIA. MON-FAH CHUNG, Arch. Derm. & Syph. **14**:111 (Aug.) 1926.

Eighty-seven patients having syphilitic disease of the spinal cord have been admitted to the Hospital of the Peking Union Medical College in the last eight years and of these, thirty-four, or 39 per cent, have had "acute" syphilitic paraplegia. The author finds this incidence high when compared with that reported recently in the Occident, but he notes that it agrees remark-

ably with the percentages reported by Spiller in 1895. He offers the suggestion that early prophylaxis, resulting from finer diagnostic measures and popular education in Europe and America, may explain the lower incidence in these continents, and that China today may correspond to Europe in the late nineteenth century.

In his study of the thirty-four cases, the author groups them clinically into three forms: (1) one showing either complete loss or reduction of all forms of sensation, and a complete motor paralysis, i. e., the clinical picture of a transverse lesion; (2) one with a dissociated sensory disturbance and paraplegia, which clinically resembles syringomyelia but in which the pathologic basis is probably thrombosis of some part of the spinal arterial system; (3) one without sensory involvement but with an affected motor function, which, except for the early acute symptoms, resembles the paralysis described by Erb. The author considers the pathologic process to be either an acute or a subacute interference with the blood supply. In the subacute cases the changes in the meninges and blood vessels correspond to those of a severe meningitis, with a rich meningeal exudate of cells and marked periarteritis, endarteritis and phlebitis, which impinges on the circulation in the small vessels about the periphery of the cord. On the other hand, he has found only thrombosis of the spinal vessels in two cases with an acute onset — in one of the dorsal group, in the other of the spinal veins — and scarcely any evidence of meningitis. Only on the basis of such a vascular lesion can the acuteness of the onset be explained.

In the acute type, of which the author has observed eight cases, there are few, if any, prodromal symptoms, the paraplegia coming on within from twenty-four to forty-eight hours and showing a complete flaccid paralysis of the lower limbs, invariably accompanied by sensory changes, loss of sphincter control and trophic disturbances. In the type with the subacute onset, of which he observed twenty-six cases, the premonitory symptoms extend over a period of about three weeks. In sixteen cases the condition began with numbness of the lower extremities; in five, with difficulty in urination and retention; in four, with weakness of the legs or difficulty in walking and, in two, with pains in the back. Usually the bladder and rectal disturbances antedate the paralysis. The sensory signs vary considerably, as would be expected, depending on the degree of vascular occlusion and the particular areas involved, syphilis not showing any predilection for any particular group of vessels.

In making the diagnosis of "acute" syphilitic paraplegia, the author wårns of the danger of overlooking a nonspecific cause of the thrombotic lesion of the cord in a syphilitic person. He does not regard the prognosis to be as unfavorable as is considered generally. Six of his patients died; seven showed a condition worse on discharge than on admission, but twenty-one showed a varying degree of improvement, in three to the point of almost complete recovery. He considers the early occurrence of twitchings and involuntary movements as favorable signs for both life and function, but when the return of sensation, reflexes, movement or the establishment of an automatic bladder does not occur for two weeks or more, he considers the outlook poor.

The author believes that recovery, if it occurs at all, takes place in spite of treatment and is the result of the establishment of a collateral circulation. The further progress of the syphilitic infection should be checked, however, by the administration of antisyphilitic medication.

PEARSON, Philadelphia.

MALARIA TREATMENT IN GENERAL PARALYSIS. DR. GERATOVITSCH, Archiv. für Psychiat. u. Neurol. **78**:64 (Sept.) 1926.

Geratovitsch reports the results of treatment with malaria and recurrent fever in general paralysis in the München Clinic during 1924. The material comprises sixty-five cases of all forms and stages of general paralysis. Fifty of these patients were treated with malaria, and fifteen with recurrent fever. The cases were taken as admitted, with the exception of those in which the general condition of the patient contraindicated high fever treatment. The material is presented under four headings. In group 1, comprising nineteen cases (29.3 per cent), are placed the patients who could return to the occupation and place in society they held before the onset of the disease; in group 2, comprising eight cases (12.4 per cent), those who could be discharged to the care of relatives as improved and capable of doing some work, but who still showed mental aberration; in group 3, comprising twenty-eight cases (43 per cent), those who could not be discharged from the hospital; in group 4, comprising ten cases (15.3 per cent), patients who died during or shortly after the treatment. The cases belonging to the first two groups are presented with concise, but fairly complete, reviews of the previous histories, physical and mental status on admission, treatment with malaria or recurrent fever, status immediately after conclusion of the treatment and immediately before discharge, and the follow-up history up to date (beginning of 1926). Of the ten cases in group 4, death in six could be traced to either intercurrent diseases or paralytic attacks, whereas in four it was definitely caused by the fever treatment.

In comparing his results with those of other authors (Nonne, Gerstmann, and others), Geratovitsch finds that the percentage of cases reported as complete remissions, i. e., belonging to the author's group 1, coincides with his own, ranging between 30 and 36. There can be no doubt as to the treatment being directly responsible for such remissions because the so-called spontaneous remissions in general paralysis never exceed 8 per cent; Kirschbaum, reporting a large number of cases, found only 1.1 per cent of spontaneous remissions. The great majority of cases treated with recurrent fever (13) belong in groups 3 and 4. The age of the patients on admission does not seem to be of great significance, but there does seem to be a direct relation between the duration of the disease and the possible improvement, for the chances of a complete remission are best when the treatment is instituted shortly after the onset. The clinical type of the disease also seems to have a direct bearing on the results, the prognosis being best in the acute "grandiose" types, and poorest in the simple dementia types. Previous treatment with specific drugs does not seem to influence the prognosis. In most cases the improvement was noticed within the first two months after the conclusion of the treatment; in some cases it was not so rapid, and others showed the first signs of improvement only five or six months later. With improvement in the mental condition, there were also some improvements in the neurologic changes, especially in disturbances of speech and writing. The treatment alsó seemed to have a great influence with patients in acute convulsions. In a few cases there was improvement also in the pupillary reactions.

Systematic study of the serologic observations during and after treatment fails to reveal any definite relation between the degree of improvement in the clinical picture and that of the serologic. Most cases of group 1, however, showed changes, the most consistent being in the cell count which was decreased. The Wassermann reaction was rendered negative more frequently in the blood than in the spinal fluid, whereas the colloidal gold and mastic reactions, although frequently changed, never reached normal.

As already noted the results with malaria were more favorable than those with recurrent fever, but it seems that only such cases were treated with the latter that for some reason showed contraindications to the former; therefore, the author does not feel that he can draw definite conclusions. Considering the fact that in three of the fifteen recurrent cases the patients developed bronchopneumonia, and also that we have no specific means of stopping the recurrent fever, one would think that treatment with malaria is the better of the two.

MALAMUD, Foxborough, Mass.

RECKLINGHAUSEN'S NEUROFIBROMATOSIS AND THE SKELETON. EDWIN P. LEHMAN, Arch. Derm. & Syph. **14**:178 (Aug.) 1926.

In 1924, the author, in collaboration with Brooks, reported seven cases of von Recklinghausen's neurofibromatosis in which marked changes in the bones were observed. At that time he classified these changes into three types: (1) scoliosis; (2) abnormalities of the growth of individual bones; (3) irregularities of the outline of bones, ranging from central and subperiosteal cysts to pedunculated subperiosteal tumors. He quotes the conclusions from his former article, that "these observations emphasize the fact that Recklinghausen's neurofibromatosis is a condition affecting bone as well as skin and nerve. The fundamental process of the disease is one of tumor growth. It has long been known that the result of this tumor growth is to produce characteristic clinical manifestations in the skin and nerves. These observations indicate that equally characteristic clinical manifestations develop in the bones. The recognition of these changes in the bones is of diagnostic importance, particularly in those instances in which the complete clinical picture, heretofore considered classical, is not developed."

In reviewing the recent literature of this condition, he expresses surprise that these observations have attracted so little attention and quotes several reports in which bony changes are mentioned but in which an understanding of the possible relationship to the disease or of its possible value in diagnosis is not evidenced.

The author reports two additional cases. In the first, there was enlargement of the right innominate bone; the femur and tibia of the right leg were slightly longer than those of the left; there was a cyst-like projection from the lateral aspect of the lower third of the left tibia, which showed the characteristic covering with cortical bone, and there was slight lower dorsal scoliosis, probably compensatory to the asymmetry of the pelvic girdle. In the second, an extremely early case showing hardly any pigmentation and tumor formation at only two sites — the cervical plexus and the cervical sympathetic chain — the author found slight scoliosis in the cervicodorsal region. The bones of the skull showed obscuration of the normal diploic markings, the texture being coarsely granular. In the right parietal region were several irregularly rarefied areas, with serrate margins, in which all bony structure was lacking.

In the light of his previous experience with bony manifestations, the author considers that these skeletal changes can be thrown into the diagnostic balance and that the second patient gradually will develop the full manifestations of the disease.

Lehman comments on three important facts, illustrated by these two cases. In the first case, overgrowth of a flat bone was found, though previously he had only observed this change in the long bones. In the second case, the reaction of the bones of the skull is manifested. In both cases, only complete

roentgenographic examination revealed the unsuspected bony involvement. The author considers that no case of von Recklinghausen's neurofibromatosis can be considered completely studied without a roentgenographic investigation of the entire skeleton.

Microscopic study of a specimen of hypertrophied bone, removed from the first patient, showed that the bone was softer than normal, being in a plastic state favorable for interstitial growth; the hard bone was diminished in quantity, and in places calcium deficiency was suggested. Is this osteoporosis due to infiltration with tumor tissue? Lehman feels that he cannot answer this question from his material, but his specimens showing the cortex penetrated by soft tissue are suggestive, at least, that this is so.

PEARSON, Philadelphia.

TUBERCULOSIS OF THE LARYNX. TREATMENT BY SURGICAL INTERVENTION IN THE SUPERIOR AND INFERIOR LARYNGEAL (RECURRENT) NERVE: A REPORT BASED ON SEVENTY-NINE CASES. HENRY P. SCHUGT, Arch. Otolaryng. **4**:479 (Dec.) 1926.

Relief of dysphagia due to tuberculosis of the larynx by nerve blocking with alcohol varies in duration, according to the statements of various authors, from a few hours to two months. All subsequent injections are less likely to be successful. In one case, the author injected both superior nerves with alcohol, giving immediate relief; forty-five minutes later, severe dyspnea necessitated an immediate tracheotomy.

In twelve cases, the author has resected the superior laryngeal nerve, with complete relief from pain in every instance. It is followed by difficulty in swallowing for a few days but "without exception there was complete and permanent relief from pain on swallowing" The operation is easily performed under local anesthesia. The inner branch of the superior laryngeal nerve was selected. In one case the hypoglossal nerve was so close to the superior laryngeal nerve that it also was blocked and the patient had paralysis of one half of the tongue. This anatomic relation is said to be found particularly in shortnecked persons.

Various authors have reported that resection of the upper laryngeal nerve has led to healing of the tuberculous process in the larynx. "In considering the question of the beneficial influence of the resection of the superior laryngeal nerve, the elimination of the sympathetic fibers within the superior laryngeal nerve must be taken into consideration." Periarterial sympathectomy for tuberculosis of the larynx has been tried. The author states that he has noticed no change in the larynx following resection of the superior laryngeal nerve except in a case, comparatively mild, in which he secured complete healing after resection. He then resected the nerve in two patients who did not have dysphagia. In one, an ulcer of the cord showed a decided tendency to heal. He concludes that resection is preferable to injection of alcohol, but "it cannot be stated that it has a direct, favorable effect on the healing of the tuberculous process."

Immobilization of the larynx has been tried by the silence treatment and tracheotomy. It may also be secured by paralyzing the recurrent laryngeal nerve, as first advocated by Leichsenring and von der Heutten. It is especially applicable to unilateral tuberculosis.

The author prefers alcohol injection of the recurrent nerve to operative exposure with freezing or cutting the nerve. He has injected the recurrent laryngeal nerve in twenty-one patients. "A needle from 6 to 8 cm. in length

is pushed along the first tracheal ring to the vertebral column, then withdrawn about 1 to 1.5 cm.; then from 1 to 1.5 cc. of an 80 per cent alcohol solution is injected. After the injection some patients complain of pain in the shoulder; this, however, disappears in a few days. The paralysis remains on an average of from about four to eight weeks, when mobility of the vocal cord returns. In my series of cases, in one case the paralysis persisted after five months, although phonation was perfectly clear." One nerve only should be injected. Objections to this method are that the side not injected does extra work, that the cough mechanism is disturbed and that expectoration is made more difficult. The author, however, feels that the method is harmless and can be repeated. In six patients he was unable to find the nerve. In three of these he made repeated injections. Of the fifteen patients in whom he succeeded in paralyzing the recurrent laryngeal nerve, nine were improved, three unimproved, two worse, and one under observation.

HUNTER, Philadelphia.

MENINGEAL HEMORRHAGE IN THE NEW-BORN. L. U. BLANCO and HUMBERTO PAPERLNI, J. A. M. A. **87**:1261 (Oct. 16) 1926.

The authors report forty-two cases of meningeal hemorrhage in the new-born. As is customary, they divide intracranial hemorrhages in the new-born into epidural, subdural, subarachnoid, intracerebral and intraventricular. Then follows a short discussion of the incidence and etiology and types of subdural and subarachnoidal hemorrhages. They divide the symptoms into two periods — excitement from intracranial pressure, and paresis or paralysis when injury to the nerve centers has occurred. There is bulging of the fontanel. They call attention to the importance of convulsions caused by hemorrhage as an aid in localization for surgery. Nystagmus, convulsion of the ocular muscles and trembling may present themselves. The authors say that continual complaint is frequently observed.

Stupor and coma, observed in many, are explained by cortical lesions. Opisthotonos, spasms and muscular rigidity with positive Kernig and Brudzinsky signs and erection of the penis are symptoms of compression of the spinal cord. Atelectasis in a robust new-born infant is claimed to be almost characteristic of a meningeal hemorrhage. Lesions of the vagus nucleus cause slow pulse followed by tachycardia. Increase in blood pressure, tense pulse, pallor and dermographism are caused by irritation of the vasomotor centers. Capillary hemorrhages are easily produced. Icterus, in the presence of other symptoms, facilitates the diagnosis of meningeal hemorrhage.

Lumbar puncture is invaluable in confirming the diagnosis, and the authors say "the application of this law (lumbar puncture in all new-born children suspected of meningeal hemorrhage) must be as constant as that of Credé in the prophylaxis of gonorrheal conjunctivitis." Prognosis has been grave, but the authors believe that early diagnosis and intensive treatment may alter this considerably. The sequelae are mental defect, spastic diplegia, hemiplegia, epilepsy, possibly internal hydrocephalus, meningo-encephalitis, sclerosis, cysts and porencephalitis. In differential diagnosis must be considered acute hydrocephalus with meningitis, meningo-encephalitis with encephalitis, concussion and intracerebral hemorrhage. Lumbar puncture, which yields a uniformly hemorrhagic or at least a xanthochromic fluid without germs, allows a differential diagnosis.

As to treatment, prophylaxis must be decided on by the obstetrician. Surgical treatment is often of avail but is attended with a mortality of 50 per cent and

does not insure always against the appearance of sequelae. In tentorial lesions, the authors recommend medical treatment by means of lumbar puncture which, in addition to its value in diagnosis, also allows of hemostatic medication at the focus of hemorrhage by the introduction of normal horse serum (from 10 to 30 cc. intraspinally) repeated several times.

Eleven cases are presented in detail. Blanco and Paperini conclude that: (1) early diagnosis and energetic treatment bring about a greater percentage of cures than was formerly believed; (2) in addition to the obstetric history one must consider asphyxia with dysphagia, bulging of the fontanel, convulsions, moaning, stupor or coma, opisthotonos, spasm and icterus; (3) lumbar puncture is necessary for diagnosis and must be done in all indicated cases; (4) in the majority of cases the treatment is medical and consists in lumbar puncture and the intraspinal injection of normal horse serum.

CHAMBERS, Syracuse, N. Y.

HEPATOLENTICULAR DEGENERATION. A CLINICAL, ANATOMIC AND EXPERIMENTAL STUDY. IVAN MAHAIM, Schweiz. Arch. f. Neurol. u. Psychiat. **16**:283, 1926.

In summarizing the subject of hepatolenticular degeneration, Mahaim stated that his purpose was to produce primary, chronic lesions of the liver and to see whether these would result in lesions of the brain. Two of five dogs operated on lived sufficiently long to fill the requirements. The first dog had received four injections of 50 per cent alcohol in 40 cc. doses through the biliary passages. He lived thirty-one days after the first intrahepatic injection without presenting any neurologic symptoms. Icterus was marked. The second dog had received two intrahepatic injections of 50 per cent ethyl alcohol. He lived seventy-nine days after the first injection without presenting any clinical symptoms. There was intense icterus and a progressive cachexia.

The livers of these animals showed marked changes of the hepatic cells with biliary pigmentation and some inflammatory reaction. The brain showed alterations of the nerve cells, with some predominance in the cortex, and vascular and glial changes of the central nuclei, chiefly of the thalamus and the caudate nucleus. These reactions were not present to any degree in the cortex. While one at first got the impression that the brain structure was uniformly involved, a closer study revealed the singular fact that the vessels of the nuclei alone were markedly damaged. The lesions of the nerve cells were regarded as a general reaction and those of the vessels and the neuroglia as more specific reactions of the central nuclei. The objection might be raised that the cerebral disturbances resulted from the absorption of alcohol rather than through the intermediate hepatic changes. Control injections eliminated this possibility. It is noteworthy that neither of the two animals developed symptoms that bore any clinical resemblance to hepatolenticular degeneration.

While the general conclusions of Kirschbaum, who also worked along these lines, did not agree with those of Mahaim, it is a striking fact that the only dog in which Kirschbaum produced primary chronic changes in the liver was the only animal that also presented distinct alterations of the central nuclei.

Mahaim concluded his series of articles with an enumeration of the following points: (1) It is legitimate, as Hall proposed, to include the three diseases, Wilson's disease, pseudosclerosis and torsion spasm, in a common group known as hepatolenticular degeneration. (2) Athetotic movements appear as a result of destructive lesions of the pallidum, although lesions elsewhere may also be present. (3) The existence of pseudosclerosis without hepatic cirrhosis has not been demonstrated. (4) Primary lesions of the liver parenchyma may be

produced by the injection of dilute alcohol into the biliary passages. (5) Primary chronic lesions of the liver of a dog produce grave generalized alterations of the nerve cells of the brain, which are a little less pronounced in the central nuclei than in the cortex. (6) Primary chronic lesions of the liver of a dog will provoke vascular and neuroglial alterations that involve the central nuclei, principally the caudate nucleus and the thalamus. Similar changes in the cortex are insignificant. They manifest themselves as early as one month after the onset of the changes in the liver. (7) These experiments make it seem probable that hepatic changes are initial in hepatolenticular degeneration. These experiments do not give any information as to the cause of the hepatic involvement.

WOLTMAN, Rochester, Minn.

THE ACUTE REGRESSIVE CHANGES OF NEUROGLIA (AMEBOID GLIA AND ACUTE SWELLING OF OLIGODENDROGLIA). WILDER PENFIELD and WILLIAM CONE, J. f. Psychol. u. Neurol. **34**:204, 1926.

Under the term neuroglia are included astrocytes and the oligodendroglia of del Rio Hortega. Microglia (del Rio Hortega's third element of the central nervous system, as well as "Gitterzellen" and "Stäbchenzellen" derived from microglia) must be clearly distinguished from neuroglia.

The term ameboid glia must be taken to signify only that astrocytes assume the shape of amebas and not that they are capable of ameboid movement. The ameboid change is a degenerative or regressive process. It is the evidence of impending cell death, and may occur diffusely through the central nervous system as an agonal or postmortem change. It also appears during continued life in localized areas. Cajal designated the same cell change as "clasmatodendrosis." This rapid change of astrocytes is a passive one and may occur in previously normal cells or in astrocytes which, owing to some chronic pathologic condition, have become hypertrophied. Such a pathologic condition may be in the nature of a chronic degeneration of nerve tissue in general and still result in an active overgrowth of astrocytes.

There is some similarity in the appearance of astrocytes, before they undergo division, and "ameboid" change of these cells. For example, the astrocytes about a recent wound of the brain may be seen to lose their fibers and swell before and during amitotic division. At this time, they resemble ameboid glia.

If either Cajal's gold chloride sublimate stain or del Rio Hortega's silver carbonate stain for astrocytes is used, two types of degeneration of astrocytes can be observed. In one, the protoplasm of the cell breaks up into discrete bodies, and there is little, if any swelling of the cell body. The bodies into which the protoplasmic expansions breaks up and the cell body are finely granular. In the second type, the cell body is swollen and its cytoplasm contains many granules. It seems that the first type is the expression of an acute degeneration, whereas in the second type the degenerative process is more gradual, permitting the cell to react in an abortive way.

In the authors' opinion, the first cell described is the type that Wohlwill, Rosental and Alzheimer, using Alzheimer's staining method V, demonstrated as containing methyl blue granules in the cell body and in the filling bodies formed by the breaking up of its projections. The second type they believe to be identical with the ameboid cell which, when stained with Alzheimer's method VI, shows fuchsinophile and lipoid cysts.

Acute swelling of oligodendroglia appears under conditions similar to those that produce ameboid glia, but earlier and often without change in the astro-

cytes. It seems probable that acute swelling of oligodendroglia will be found in a number of types of toxic deliriums and stupors. The study of these cells, however, necessitates the removal of tissue by operation or by early necropsy.

The original article is in English, and is unusually well illustrated.

KESCHNER, New York.

PROGRESSIVE MUSCULAR DYSTROPHY WITH EXTRAPYRAMIDAL SIGNS. A. WESTPHAL, Klin. Wchnschr. **5**:1404 (July 30) 1926.

Westphal gives the clinical report of a case of progressive muscular atrophy associated with extrapyramidal signs. The patient was a man, aged 25, first examined by him in October, 1925. The family history was unimportant. In 1918, the patient first noted beginning weakness and atrophy of the muscles of the left shoulder girdle and arm. By 1919, the process had extended to the right shoulder and arm. The examination in October, 1925, showed a marked lordosis and extensive atrophy of both shoulder girdles and arms with corresponding weakness. In the forearms the only muscular involvement was that of the supinator longus. The left abdominal muscles also showed slight weakness. The remaining musculature did not show any change. There was no evidence of hypertrophy. Fibrillary twitchings were absent, but marked choreiform and myoclonic twitchings of the atrophic muscles were observed. These movements were of a complex character, at times rhythmic and again arrhythmic. The only atrophic muscles that did not show any abnormal movements were the supinator longus muscles. No twitchings were noted in the normal musculature. Beyond these observations, the general and neurologic examinations gave negative results.

In the discussion, Westphal states that one is dealing with a typical juvenile progressive atrophy, associated with an extrapyramidal disturbance. The localization of the pathologic movements is most unusual. It seems impossible to explain the observations on the basis of a heredodegenerative process. It was also impossible to explain these symptoms on the basis of an infectious process as there had not been any evidence of such a disturbance, though it is recognized that such residuals may occur even though a history of an acute process is not obtained. It is the author's belief that one may be dealing with an affection of the interbrain, basal ganglia or substantia nigra. To assume that the present case has its pathologic change in this region is, of course, unwise, as pathologic studies have not been at all conclusive.

Choreic symptoms have rarely been described in progressive muscular dystrophy. Erb mentioned such an occurrence and also refers to the case of Le Noir and Besancon. Frau Schultz refers to a case of infantile progressive dystrophy with atrophy of the bones and marked increase of sexual characteristics. This is rather the contrary to the postencephalitic cases of dystrophia adiposogenitalis with hypoplasia of the genital organs. The association of progressive muscular dystrophy with polyuria and diabetes insipidus has been described frequently, and it has been assumed by some that the lesion is in the floor of the third ventricle. Westphal cites other cases, trying to point out the possibility of some localized pathologic change in the interbrain to account for these unusual associated symptoms but unfortunately, as is so often true, definite conclusions cannot be drawn. The author does not mention the association of progressive muscular atrophies with such degenerative processes as Friedreich's ataxia which occasionally have been seen and reported in the literature.

MOERSCH, Rochester, Minn.

RELATIONSHIPS OF EPIDEMIC ENCEPHALITIS TO SOME HYSTERICAL MANIFESTATIONS. C. MARINESCO and A. RADOVICI, J. neurol. et psychiat. **26**:259 (May) 1926.

Study of some hysterical manifestations following encephalitis led the authors to discuss the relationship between encephalitis and hysteria. The most striking manifestation studied was the appearance, in a series of ten cases, of periodic contractions, tonic in type and involving especially the oculomotor muscles. During the fit the patient showed conjugate deviation of the head and eyes with forced rotation of the head and even of the trunk. An organic substratum of the manifestations is maintained because of the presence of vestibular disturbances, disturbances of the vegetative nervous reflexes, especially the oculocardiac reflex, and because atropine and scopolamine instantly stopped the fit. Special attention is paid to some factors that were supposed to start the fit — fatigue, emotional factors and the action of suggestion by simply asking the patient to assume a position of the head and eyes similar to that shown during the fit. The authors even succeeded, in some instances, in stopping the fit by therapeutic persuasion, as, for instance, by the injection of simple distilled water. Finally, the fits were sometimes accompanied by other hysterical manifestations such as rigidity of the lower extremities or passional attitudes of the whole body.

The authors are inclined to explain these manifestations as the result of disturbed control of the cortex over the subcortical centers owing to a lesion of the basal ganglia and of the hypothalamic region. The inhibitory fibers being partially destroyed, the external impressions reaching the subcortical centers are then transformed into the simple movement of imitation. The encephalitic lesions of the basal ganglia may then be considered as the anatomic basis of the psychotic disturbances and of the abnormal suggestibility. The presence of evident signs of vagotonia during the fit leads, however, to the impression that the vegetative nervous system may to some extent form a background, one influencing the humoral conditions of the organism as shown by dysequilibrium of the vegetative nervous system as well as by disturbance of the acid-base equilibrium found in postencephalitis.

Whatever the mechanism of the action of the suggestions is, the authors consider it the result of the functional condition of the different organs. They do not consider a phenomenon of escape from any determining factors, but the expression of a complete dynamic condition the nature of which may some day be explained.

FERRARO, New York.

HYPERREFLEXIA OF LOWER LIMBS AFTER EXERCISE. W. G. SPILLER, J. A. M. A. **87**:639 (Aug. 28) 1926.

Spiller emphasizes the fact that a distinction must be made between the effect of prolonged exercise and that after moderate exercise for only a few minutes. Lombard found that moderate fatigue produced a decided diminution in the knee reflex. Most authors believe that the reflexes are exaggerated after moderate fatigue, while excessive fatigue abolishes or greatly diminishes them.

Spiller reports the case of a man, aged 29, who while at Camp Green, N. C., during the late war, awoke one morning to find both lower limbs powerless. Any long hike for weeks previous had caused the limbs to feel numb and weak. After a few weeks without any further subjective symptoms he returned to duty and was found to be normal, except for a numb feeling in the left lower

limb after long hikes. He saw service overseas, and although he complained of discomfort in his left lower limb during his entire stay in France, he was discharged in 1919 without disability. In May, 1921, Spiller's examination disclosed markedly exaggerated patellar reflexes, bilateral patellar clonus, persistent left ankle clonus, and probable bilateral Babinski sign. There were no abnormalities other than motor weakness of the left lower limb; sensory examination, lumbar puncture and the roentgen ray revealed nothing abnormal. Spiller believes that organic change had occurred in the spinal cord probably in relation to excessive exercise.

Spiller reports a case of hyperreflexia after slight exercise and believes that this is a rare occurrence, indicating perhaps a beginning degeneration of the pyramidal tracts. In a man, aged 51, the tendon reflexes of the lower limbs were greatly exaggerated (he had walked several squares). He was sent to the hospital, where the intern's examination did not reveal any abnormality except some exaggeration of the right patellar reflex. This caused Spiller to believe that a mistake had been made, and he confirmed the observations of the intern; a little exercise, however, caused great increase in the reflex activity of the lower limbs, while in the morning, after rest, this had greatly subsided. Exercise produced bilateral clonus. The plantar reflex was always flexion. There was no spasticity nor paresis. The serology was normal. Spiller regards this as possibly a case of incipient spastic paralysis. Rouquier and Couretas report a case of syringomyelia with somewhat similar characteristics, but no mention of such a condition in any other disease has been made, according to Miller.

CHAMBERS, Syracuse, N. Y.

CONSTITUTION AND PSYCHOSIS. G. VERMEYLE, J. neurol. et psychiat. **25**:297 (May) 1926.

The author deplores the tendency of some observers to go too far with the conception of the psychopathic constitution and to consider the psychosis only as a constitutional disease ruled by primitive morbid tendencies. The author believes that the relationship between the constitution and psychosis is not so simple; psychoses are often observed in which the previous personality of the patient plays no part. He divides the psychoses into two main categories: (*a*) The idiopathic or constitutional psychoses which are exaggerations of the patients' habitual condition. In these psychoses, internal and external influences play a small part in the appearance, development or disappearance of the psychosis. The onset as well as the recovery is as a rule almost abrupt, no incubation period or period of convalescence being noticed — recurrence is almost the rule. (*b*) The symptomatic psychoses, which constitute a new fact in the psychophysiologic development of the patient. They are not a direct result of his previous personality, but are a real mutation, a deep transformation of his physical and psychic condition. The transformation in this case is gradual and slow, due to toxic or infectious factors as well as to the organic disturbances in the functioning of the digestive circulatory, respiratory or neuroglandular apparatus. At other times emotional factors, with the humoral changes that they produce, may exert morbid influence. Recovery in these symptomatic psychoses is not rapid nor abrupt but is the result of a gradual return to the previous physiologic equilibrium. Natural tendencies have little or nothing to do with the clinical manifestations. However, in some of these psychoses, regressive tendencies, in the meaning of Mendel, may have some influence.

The importance of studying the two categories lies in prognosis, which varies in the two groups. Indeed, while in an idiopathic psychosis treatment is really ineffective, the psychosis being ruled by internal factors which unfortunately are often intangible, there is a larger field of action in the symptomatic psychosis and results are obtained by psychotherapy or by medicinal measures.

FERRARO, New York.

HISTOCHEMICAL STUDY OF IRON COMPOUNDS IN THE CEREBRAL AND CEREBELLAR CORTEX OF THE INSANE. C. TRÉTIAKOFF and CAESAR OSORIO, Rev. neurol. **2**:220, 1926.

It has only recently been recognized that iron is present in the higher nerve centers of man, and its significance is not well understood. The amount probably is small, and in the brain it probably acts as a metal catalyst, regulating metabolism. It is probably present in something less than 1 part in 10,000. The demonstration of iron in the brain is rendered difficult because in its organic compounds it resists the ordinary chemical tests.

The authors studied the reaction in the gross by the following method: Sections from 1 to 2 cm. thick were fixed in 95 per cent alcohol for about a week, and then rapidly embedded in celloidin. Sections from 2 to 5 mm. thick were cut by a macrotome. To remove the fatty substances the sections were washed for five minutes each in ether, absolute alcohol, and lower concentrations, finally going into distilled water for from fifteen to twenty minutes. The sections were then carried into a freshly made saturated aqueous solution of ammonium sulphide. After a few minutes a slight greenish blue color became visible in regions richest in iron. The reaction took about two hours, and the result was seen as a dark bluish green color in those parts containing the iron. The sections were best examined in the reagent, because distilled water weakened the color. The sections could be kept for a long time in the reagent if the flasks were well stoppered. This method proved more satisfactory than the sulphocyanate or ferrocyanide method.

The results obtained by the authors were as follows: Dementia paralytica gave an intense coloration. Epilepsy, alcoholism, and imbecility showed a weak reaction, and brains from manic-depressive, dementia praecox, and paranoid patients gave a variable reaction. The staining was variable from case to case and from place to place in the brain. Perhaps the most characteristic location was the frontal lobe.

FREEMAN, Washington, D. C.

EPENDYMAL GLIOMA: A NEUROLOGIC STUDY; STUDY OF THE VESTIBULAR FUNCTIONS. BUYS P. MARTIN and L. VAN BOGAERT, J. neurol. et psychiat. **25**:203 (April) 1926.

The authors describe a case of ependymal glioma of the cerebellum in which the operation was successful. From the clinical point of view, there was an evident cerebellar syndrome characterized by spontaneous deviation of the body toward the left side, slight hypotonia of the left side and hyperactivity of the deep reflexes of the upper extremities, all signs suggestive of a localization in the left side of the cerebellum. A point of special interest was the presence of urogenital disturbances characterized by an early lack of "ejaculatio" and later by absence of erection and difficulty in urination. The vestibular reaction was tested, and three main points were observed: (*a*) spontaneous nystagmus toward both the right and the left side; (*b*) presence of the so-called postural nystagmus, viz., left nystagmus elicited by bending the head

backward and toward the right, bending of the head toward the left side being without response; (*c*) deviation of the right arm toward the right, which accompanied the nystagmus. All these signs are suggestive of a peripheral involvement of the vestibular nerve.

The patient was operated on and the tumor, which macroscopically recalled a glioma, was found to occupy the midline of the cerebellum backward to the floor of the fourth ventricle. The tumor did not develop from the cerebellum itself and only compressed that structure. Owing to its prolongation into the ventricle, the tumor was only partially removed. Histologic study revealed an ependymoglioma, a rare form of tumor that recently has been carefully reviewed by Cushing and Bailey. Although the course of this type of tumor is generally fatal, the patient recovered from the operation and four months later presented only a few slight signs of the previous neurologic condition.

FERRARO, New York.

THE STRUCTURE AND FUNCTIONS OF THE ACOUSTIC SYSTEM IN THE WHALE. WALTHER RIESE, J. f. Psychol. u. Neurol. **34**:194, 1926.

It is not yet definitely known how the whale reacts to auditory stimuli, although comparative anatomy has clearly established that the central cochlear apparatus in this animal is unusually well developed, though it differs greatly from the acoustic system in many of the other mammals—higher as well as lower. Jelgersma assumes that in the whale the vestibular stimuli are registered in the cochlear system; this assumption is based on two hypotheses: (1) the cochlear system in this animal is atrophied at the expense of the vestibular system; (2) the cochlear elements released by this atrophy assume a preponderance of vestibular function. Whether or not the two hypotheses are absolutely correct cannot, in the present state of knowledge, be determined, but one thing is certain, and that is that one cannot look for a "gnostic" faculty for auditory impressions in the whale. The definitely limited internal geniculate body in this animal would seem to be against the conception of a cortical connection with that part of the brain which in other animals is ordinarily associated with the center for audition. In the whale, cochlear impulses are to a great extent, if not exclusively, converted into certain motor reactions which are necessary for the animal's "protection" ("reflexes for defense," "flight reactions," etc.), and which are of such vital importance to aquatic animals in general. There is ample reason to believe that, analogously to the optic system, the acoustic system contains acousticognostic and acousticostatic components which are in a measure independent and separate systems. As a matter of fact, it would not be inconceivable to seek the anatomic basis for the acousticostatic elements in the deeper acoustic centers of the interbrain and midbrain.

KESCHNER, New York.

Society Transactions

SOCIÉTÉ DE NEUROLOGIE DE PARIS

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ANDRÉ LÉRI, M.D., and G. ROUSSY, M.D., *Presiding*

Rev. neurol. **1**:50, 1927

SYNDROME OF THE LOWER RED NUCLEUS. DRS. CLAUDE GAUTIER and JEAN LEREBOULLET.

A patient presented alternate hemiplegia, characterized by paralysis of the third nerve on one side and of the cerebellar syndrome on the other, but without motor or sensory disturbance. The case was almost identical with one reported in 1912 by Claude, even in presenting disorders of speech and in the absence of facial paralysis. In this case the condition was probably not total, but partial, paralysis of the red nucleus, involving only its inferior portion. This syndrome should be distinguished from the syndrome of lesion of the upper portion of the red nucleus, which gives rise, in addition to cerebellar disorders, to certain thalamic symptoms, and yet does not result in oculomotor paralysis.

The paralysis of the third nerve was complete on the right side. On the left side there was paralysis of upward movement. The hypothesis of a nuclear lesion of the third nerve on the left side, affecting the right superior rectus and superior oblique, was not probable, because the blood vessels do not cross the median line. The hypothesis of ischemia or edema from compression of the side opposite the principal lesion was also unlikely. It seemed, therefore, to be paralysis of upward dissociated movement, known as "Parinaud's syndrome," although in this case convergence was maintained.

PARINAUD'S SYNDROME AND DOUBLE CROSSED SYNDROME OF FOVILLE WITH HEMIPLEGIA OF ENCEPHALITIC ORIGIN. DRS. LAIGNEL-LAVASTINE and BOURGEOIS.

A woman, aged 30, entered the hospital in October, 1926, on account of a left hemiplegia which had begun six months before. The disorder progressed slowly, but in September there was a sudden advance of the paralysis, without a stroke, and the patient had to go to bed. The motor disturbances were accompanied by intense paroxysmal headache without special localization. The patient had lost weight, had a sleepy feeling during the day and nocturnal insomnia, but had no fever. Four or five months before the onset of the disease the patient had had otitis on the left side, but there were no residuals.

On examination there was hemiparesis of the lower portion of the left side of the face. All movements were possible, but weak. Walking was also possible, but was rendered difficult by dizziness. The tendon reflexes were lively on the left side, but there was no clonus. The Babinski sign was present on the left side. Sensibility was not affected in any way, nor were there sympathetic disorders. Examination of the eyes showed, on the right side, paralysis of lateral movements to the outer side, with concomitant diminution of the excursion of the left eye looking in. Nevertheless, there was diplopia in the

extreme right lateral deviation due to more marked paralysis of the right rectus externus than of the right rectus internus. The pupils were equal and reacted normally. There was no hemianopia. The patient suffered from dizziness, especially on standing, with lateropulsion to the left, together with nystagmus and dysmetria and adiadokokinesis on the left side. Lumbar puncture yielded normal results.

When moved to the ophthalmic clinic for examination, the patient had a stroke, which was followed by coma lasting several hours. After this the following observations were made: Complete hemiplegia with marked contracture was present on the right side. The Babinski sign was positive on both sides, and the lower portion of the face was affected. The pharyngeal reflex was abolished. Conjugate movement of the eyeballs to the left, which was possible before the accident, was abolished. Movements of elevation and depression of the eyes were lost for a few hours following the stroke, but reappeared. There was no paralysis of convergence. Lumbar puncture at this time yielded xanthochromic fluid, indicating meningeal hemorrhage. The hemiplegia cleared up rapidly, and the conjugate movements to the left returned, but there was still nystagmus on looking to the left. Following this the left hemiparesis became less and is now almost absent. The Babinski signs have disappeared. The tendon reflexes are about equal. Voluntary movements of the face are normal, although the aspect is somewhat washed out. Deviation of the eyes to the right is still limited, but there is no diplopia.

With a negative Wassermann reaction throughout and a normal fluid pressure, the presence of these focal signs recalls the Wernicke type of encephalitis, and suggests a relationship to epidemic encephalitis. Such hemorrhages may occur in ordinary encephalitis.

RECURRENCE OF CEREBROSPINAL MENINGITIS A YEAR AFTER THE FIRST ATTACK. DRS. LENOBLE and HOULLIER.

A young woman had an attack of cerebrospinal meningitis in October, 1925, with recovery in about ten weeks following intraspinal injections of serum. She felt considerable fatigue in walking. Almost a year later she complained of pains in the heel, forcing her to sit down, but, except on movement, she felt no pain. There was no fever and no cutaneous eruption. The tendon reflexes were normal, and there was no Babinski or Kernig sign. The patient complained of headache. There was no disturbance of the pupils and no disturbance of sight or hearing. The pressure of the cerebrospinal fluid was elevated. The fluid showed 77 cells and fairly large amounts of albumin, with no sugar. Smears were negative, but the culture yielded a staphylococcus with numerous other bacteria, probably contaminations. Antimeningococcic serum was administered intraspinally and subcutaneously. The lymph nodes became swollen, and there was generalized urticaria that yielded to treatment with calcium chloride. She received altogether 240 cc. of serum.

TUMOR OF THE CEREBELLOPONTILE ANGLE (WITH FEW PHYSICAL SIGNS AND PRONOUNCED DEMENTIA). DR. B. CONOS.

A woman, aged 55, entered the hospital on account of weakness in walking and severe dementia of simple type. The patient was disoriented in space and time, could not name her children or even remember how many she had. She complained of confusion. She was incontinent and indifferent, but uncomplaining. The pupils were equal, reacted well to light and in accommodation. There were certain horizontal nystagmoid movements. Speech was normal.

The tendon reflexes of the upper extremity were lively, those of the lower extremities more marked on the left side; there was no clonus or Babinski sign; the abdominal reflexes were not obtained. Muscular power was somewhat less on the right side. Sensibility appeared normal. The patient could stand by spreading the legs, but walking was impossible. The spinal fluid was clear, with a negative Wassermann reaction and no cells. The glucose was 125 mg. and the albumin 78 mg. per hundred cubic centimeters.

Some months after admission the right pupil was noted to be larger than the left, and there was weakness of the external rectus muscle on both sides. There were nystagmoid movements, especially on looking to the right. The face was not paralyzed. The patient could not lift the right leg from the bed without flexing the various segments. There was little incoordination in the finger-to-nose test, although the patient's dementia was too great to enable her to carry out tests accurately. The incoordination was more marked on the right side. The patient did not complain of headache. The eyegrounds were not examined. The diagnosis was multiple areas of thrombotic softening, although toward the end a cerebellar tumor was considered.

The patient died after twenty-four hours in coma. A tumor the size of a plum was found in the left cerebellopontile angle, apparently growing from the medulla and presenting certain segmentation resembling cerebellar tissue. Histologically, the diagnosis was fibrosarcoma. Although the presence of severe dementia rendered the diagnosis difficult in this case, there were a few neurologic symptoms that could be elicited on clinical examination. The absence of facial paralysis was noted. Attention was not drawn to the hearing. There was no headache or other sign of pressure, and only in the last weeks of life was weakness of the external recti noted. The disturbance of coordination was considered possibly to be of cerebellar origin. The albuminocytologic dissociation in the cerebrospinal fluid is worth noting. It is found in cases of tumor of the brain, especially in tumors at the cerebellopontile angle.

THREE CASES OF TRAUMATIC NEUROSYPHILIS. DRS. URECHIA and MIHALESCU.

Localization of disease at the locus minoris resistentiae seems to hold good for syphilis. Nevertheless, cases of traumatic neurosyphilis are rare, and many of these are doubtful. In these three cases the relationship between the traumatism and tabes, hemiplegia and epilepsy seems evident.

CASE 1.—A brakeman, aged 36, in 1925, was struck on the left side of the head, above and behind the ear. He lost consciousness. The next morning he noticed a small mass in this region, slightly painful. It broke down and ulcerated in a month. At the same time that this gumma softened, two others appeared on the right side of the head. About six weeks after the injury, and progressing simultaneously with the gummas, hemiparesis developed on the left side. There were fairly marked constitutional signs of syphilis, and the serologic reactions were all positive. The pupils were unequal and irregular, with diminished response to light. The tendon reflexes were increased on the left side with ankle clonus but with a negative Babinski sign. There was some spasticity. Roentgenograms showed the shadow of an osseous gumma in the left temporal region, over the area of trauma. During the antisyphilitic treatment the patient had a convulsion, but within two months the gummas and evidence of hemiparesis disappeared.

CASE 2.—A man, aged 44, had had no condition that might have been thought to be tabes, although he had contracted syphilis nineteen years before. In a railroad wreck he had suffered a violent traumatism in the lower part of the

back and a fracture of the left arm. Following this accident, there were pains and paresthesias in the feet, with disturbances of gait. Physical examination revealed somatic signs of syphilis, together with anisocoria and abolition of tendon reflexes in the lower extremities. The lancinating pains also affected the upper extremities. Gastric crises were present. There was some improvement under antisyphilitic therapy.

Case 3.—The patient had a syphilitic infection in 1913; in 1918, during the war he was struck in the left parietal region and lost consciousness. Ten months later, infrequent attacks of epilepsy occurred, which diminished in frequency until 1925, when headache and more frequent attacks developed, and the patient submitted to exploratory operation which did not result in any improvement. The attacks were preceded by a sensory aura, and were practically limited to the right half of the body. Somatic signs of syphilis were present with defects in bone and positive serologic evidence of syphilis. The patient improved under antisyphilitic treatment, and the attacks became less frequent.

A Case of Priapism. Drs. Conos, Zacar and Manouelian.

A man, aged 35, married for eight years, said that he was not addicted to alcohol, had not had syphilis and did not practice masturbation. Beginning in November, 1926, he noticed gradual reduction in potency and took a preparation of zinc phosphate and strychnine for ten days. Ten days after he stopped taking the drug, on waking from a passionate dream, he had a strong erection, which neither injection of morphine nor cocainization of the urethra reduced. On the recommendation of a friend, he had sexual intercourse three times, but in spite of ejaculations, the erection did not relax for a moment. No medication was effective. Chloroform reduced it only temporarily during the narcosis. After four weeks of ineffective treatment, he entered the hospital, still with strong erection and severe pain. There was no fever; the pulse was rapid; blood pressure was normal. The penis was in extension practically along the abdominal wall, somewhat deviated to the left, cyanotic, hard and tender. The scrotum contained normal testicles with normal sensibility. The patellar and achilles reflexes were lively, more so on the left side. The plantar reflex was indifferent. The abdominal reflexes were normal. The cremasteric was more lively on the right side. Muscular power seemed somewhat diminished in the lower limbs. The sphincters functioned well. The patient was in poor condition. Many different medicines failed to have any effect. Lumbar puncture revealed clear fluid under normal pressure, with a negative Wassermann reaction. An intraspinal injection was then given consisting of 4 cc. of 1 per cent procaine hydrochloride with 10 drops of epinephrine. Three fourths of an hour later, the patient went into syncope with profuse sweats and thready pulse, successfully treated by stimulation. The erection was noticeably diminished during the syncope, and following this it gradually became reduced. The patient left the hospital five days after the lumbar puncture, and apparently was completely cured.

The patient presented no signs of general disease, such as leukemia, uremia, hydrophobia or encephalitis, of a spinal disease, such as multiple sclerosis or tabies, of disease of the spinal column such as traumatism, tumor or tuberculosis, nor of a local disease of the genitals. The character of the reflexes suggests some involvement of the spinal centers, and it is natural to suppose that the aphrodisiac brought about hyperemia of the lumbosacral region of the cord. The painful erection lasted for forty-five days, which is not unusual;

cases have varied in duration from two days to two years, and one author reports priapism as a predominant symptom in multiple sclerosis for eighteen years.

PINEAL TUMOR TREATED BY THE ROENTGEN RAY. DRS. ALAJOUANINE and GILBERT.

Cases of successful roentgen-ray therapy for tumor of the brain are always worth reporting. This patient was presented fourteen months before, with a syndrome of intracranial hypertension and intense headache, vertigo and stupor, attacks of narcolepsy and later diplopia with a noticeable difficulty in looking up and down. In September, 1924, paralysis of vertical conjugate movements, of convergence and of divergence was noted, with crossed diplopia in looking straight ahead and a homonymous diplopia on lateral movements. A typical Argyll Robertson sign was present. Visual acuity was reduced, and on ophthalmoscopic examination choked disks with venous hemorrhages were seen. The patient complained of pulsating noises in the ears and painful hyperacusia, but the labyrinthine examination gave normal results. There was moderate incoordination, hypotonia and abolition of postural reflexes. Painful sensations were aroused by stimulation of the right side of the body. There were dissociated thermic hypesthesia, vasomotor changes and corneal anesthesia on the right side. The spinal fluid was under considerable pressure, with some lymphocytosis and hyperalbuminosis. The Wassermann reaction was negative. A roentgenogram of the skull showed a dense shadow, as large as a pea, corresponding to the pineal gland. Bilateral temporal decompression was performed without reduction of the headache, and the symptoms in general progressed rapidly until a typical bilateral cerebellar syndrome was noted. The localization of the tumor in the region of the corpora quadrigemina was not difficult. Although marked calcification of the pineal occurs in about 1 per cent of patients, its association with symptoms in the adjacent region allows one to say only that the tumor occupies the quadrigeminal region.

Roentgen-ray therapy was then undertaken, with four aspects and in the usual doses. The series was repeated four months later. During the first month of the first series, the patient had no more headache, somnolence or narcolepsy. The auditory phenomena and the pain on the right side of the body had disappeared, and vision became normal. Fourteen months later there was normal visual acuity, with complete disappearance of the choked disk. There was no diplopia and no weakness in divergence, but the syndrome of Parinaud and the sign of Argyll Robertson remained. Careful examination failed to disclose cerebellar or sensory disorders. This rapid improvement has been maintained.

FREEMAN, Washington.

CHICAGO NEUROLOGICAL SOCIETY IN JOINT MEETING WITH CHICAGO ORTHOPEDIC CLUB

Jan. 20, 1927

JOHN FAVILL, M.D., *in the Chair*

SCIATIC NEURITIS. DR. HUGH T. PATRICK.

Many years ago, that remarkable observer and erudite physician, Jonathan Hutchinson, asserted that a certain number of cases of sciatic pain were due to trouble in the hip joint. About forty years ago, Dr. L. Putzel of New York called my attention to the fact that chronic arthritis of the hip was often mis-

taken for sciatica. In 1913, the elder Bruce vociferously revolted against the idea that sciatica is a common disease and seemed inclined to believe that all cases of sciatica were really cases of arthritis of the hip joint. About ten years ago, I published a paper on brachial neuritis and sciatica, which seems to have made remarkably little impression.

My thesis is simple, viz., sciatica is a rare and unusual disease. The general medical public is not aware of this fact. Consequently, of the total cases apparently diagnosed of sciatica, roughly fully 90 per cent are arthritis of the hip or spine, trouble in the sacro-iliac joint, sacralization of the lumber vertebrae or other anomalous growth or formation of the lower spine, spinal caries, malignant or benign neoplasm of the spine or pelvis, intrapelvic disease of some sort, osteomyelitis or periostitis, syphilis or tumor of the lower cord or cauda equina, tuberculous hip, an extraspinal something pressing on or irritating the sacral plexus or sciatic nerve, tabes, or syringomyelia. Doubtless there are other things that may simulate sciatica.

In my private work, I see perhaps one case a year of what looks like real sciatica. Even then I feel uncertain about it unless I can follow the case for some time. The number of cases of pseudosciatica, in which a diagnosis of sciatica has been made or which were referred to me as sciatica, is not easy to estimate without minute scrutiny of all my case records, for all such cases are not indexed under sciatica. For instance, here is a case referred by a reliable physician as one of sciatica. It proved to be cancer of the lower end of the spine. Another, referred by one of the best surgeons of Chicago who said that careful examination, including roentgenograms, showed nothing wrong with the hip, was a clear case of arthritis of that joint. It is fair to say that for one case of sciatica I see fifteen falsely supposed to be that disease. Presumably no one would come to me or be sent to me for arthritis, rheumatism, tumor of the bone, etc.

Frequently I have been told that at the Cook County Hospital real sciatica is not at all rare. It is interesting to note that my associate, Dr. Levy, during three months on the neurologic service at the Cook County Hospital, saw seven patients admitted with the diagnosis of sciatica made in the receiving ward. Of these, the diagnosis was confirmed in only one. Three of the patients had arthritis of the hip, one myositis, one tabes and one cerebrospinal syphilis. And I allow myself some doubt as to the seventh case.

To speak of differential diagnosis before this audience seems like an affront, but I may mention a few points: (1) Negative roentgenograms mean little or nothing. (2) Sciatica causes no limitation of motion except such as involve stretching of the nerve or roots of the plexus—the Lasègue sign—and this sign may be present in involvement of the nerve roots by the arthritic process in the spine, syphilis, etc. (3) In arthritis of the hip, there may be no apparent limitation of motion. One must examine for it. The various movements at the hip should be executed *against resistance*. What I call the "hip sign" is, I think, the most delicate test for arthritis of this joint. With the patient supine, the ankle or outer malleolus of one side is placed above the patella of the opposite fully extended leg and then the knee of the side being examined is depressed toward the table or couch on which the patient is lying. If the hip joint is normal, even in old people, the knee can be depressed easily and without pain until the leg is almost horizontal. Of course in carrying out this test, the pelvis must not be tipped. If the arthritis is active, this movement in painful. If there are simply the remnants of an old arthritis which is not active, there will be resistance to the depression of the knee without pain. In many

of the chronic or subacute cases of arthritis of the hip, the thigh can be flexed on the abdomen, and it can be abducted to practically the normal extent; ordinarily, rotation is not painful, and driving the head of the femur into the acetabulum by forcibly striking the sole of the foot or by heavy percussion over the trochanter may be painless, but this hip sign will show trouble in the joint. 4. So-called tender points are a delusion and a snare. They are of practically no diagnostic value. 5. Sometimes the state of the deep reflexes excludes sciatica at once. In arthritis of the hip joint the knee reflex is nearly always exaggerated and frequently the achilles reflex as well. In sciatica, the achilles reflex is diminished or lost. 6. In arthritis of the spine, the achilles reflex may be diminished, and there may be some anesthesia and some muscular wasting. 7. In arthritis of the hip, there is nearly always some wasting of the buttocks and sometimes of the thigh, and if the knee joint is involved there may be wasting of the muscles of the leg. (8) Careful inquiry and examination will frequently reveal involvement of other joints in cases of pseudosciatica. (9) The presence of fever and leukocytosis enables one at once to make a diagnosis of pseudosciatica. (10) The explanation of why the scoliosis of "sciatica" is sometimes toward the affected side and sometimes away from it has been well explained by Sicard and his pupils in their careful and extensive studies of arthritis of the spine and the pathologic relations of the intervertebral foramina.

DISCUSSION

Dr. John Ridlon: I fully agree with Dr. Patrick and wish to emphasize that arthritis may exist with a negative roentgenogram; an arthritis that does not show in the roentgenogram may be the cause of more tender joints than one that does show. For the instruction of the neurologists present, I will tell you how to cure all cases of sciatica that are not due to the causes Dr. Patrick mentioned. All true sciatica is due to slow acting bowels, and is cured by senna tea.

Orthopedic Treatment of Infantile Paralysis. Dr. Edwin W. Ryerson.

When the acute symptoms have subsided, braces or molded plaster of Paris posterior splints should be applied to the feet and legs to prevent the feet from assuming a position of deformity and the knees from becoming permanently flexed. The thighs must not be allowed to remain abducted, as otherwise a contracture of the tensor fasciae latae will occur.

When the painful stage is passed, gentle massage and passive movements of all joints are practiced daily, and the patient is encouraged to attempt to use the paralyzed muscles. Systematic muscle training is of great importance, both now and later. Absolute recumbency in bed for many months is advisable in the severer cases. Electric treatment is of doubtful value, but can do no harm. After the period of recumbency, the patient can gradually assume the upright position, protected from deformity by casts or braces, which should include the torso if any paralysis of the abdominal or back muscles has been discovered. The muscle training must be continued for two or three years and a careful survey then made of the residual paralysis. In many cases there is weakness of some of the leg muscles, most often the extensors of the foot and thigh, but frequently involving other groups. In cases of extensive paralysis, braces, crutches, or wheeled chairs may be the only possible relief, but in the ordinary cases it is possible to stabilize the feet and knees by operative procedures so that walking without external aid can be performed. The most widely useful operation on paralyzed feet is undoubtedly the Whitman astragalectomy with back-

ward displacement. Most other methods are too highly technical for discussion in a meeting of this kind. Tendon transplantations at the knees are often useful.

Paralytic scoliosis is extremely difficult to control; many of the more radical orthopedic surgeons believe that operation in such a case offers the greatest hope. After the spine has been straightened as much as possible by traction in bed on a curved frame, the vertebrae over the involved area are ankylosed together by an ingenious and complicated operation, thus preventing the increase of a deformity, which under any other known method of treatment would be certain to increase.

DISCUSSION

Dr. Hugh T. Patrick: I wish to mention an illuminating case, seen many years ago in Paris. A girl, aged 12 or 14 years, had had acute poliomyelitis at 1½ or 2 years of age, which involved all extremities. From the time of onset the child had had absolutely no treatment until about a year before I saw her. She had been allowed to lie flat on a cot, completely quadriplegic. She was fed, and that was all that was done for her. Finally she was brought to the clinic of Berrillon, a sort of semiquack, who indulged in therapeutic methods which were not current. He hypnotized this child regularly, but at the same time used massage and electricity of various kinds. The child improved so that she not only could stand after having been absolutely helpless for ten years or more, but also could walk fairly well and had considerable use of the arms. Obviously, some muscles had not improved and never would. Certain cells in the spinal cord had recovered, but as no effort had been made to move the muscles supplied by these cells, they remained paralyzed until she received some mechanotherapy. The girl looked to me as if she was in about the same condition she would have been if treatment had been begun at once and continued during the ten years.

Dr. John L. Porter: One feature that I think should be recognized and emphasized is the necessity for prolonged rest after the initial stage. I wish to call particular attention to one feature that must be combatted in securing prolonged rest for many of these children. Several years ago I was called to see a patient with infantile paralysis within a week of the onset. The involvement of the tibialis and the toe-drop were noticeable. The child had just recovered from the high fever. The mother was holding the child in her lap, and when the physician called attention to the paralysis and toe-drop the mother said the child had no paralysis and instructed the child to get down and walk to show us that walking was possible. In spite of all we could do, that child was up and dressed every day and was made to walk. The pathetic part was that the paralysis progressed for a year. The tendency of parents to fail to acknowledge the presence of paralysis is a factor in the lack of rest.

Dr. Ryerson: In regard to Dr. Patrick's interesting remarks about the case in Paris, I think that child had sufficient rest. It may be that we are not giving these children rest enough, and that if we allowed them to lie in bed for ten years they would do better. I agree with Dr. Porter that all the parents want their children to get up and walk as soon as possible, but according to the views of all of us, except Feiss of Cleveland, we should urge complete rest for a long time. The trouble with infantile paralysis is that each case is different. Many cases of infantile paralysis, which not only are not recognized but also are absolutely unrecognizable, undoubtedly occur every year. There is a definite anterior poliomyelitis in many cases with no paralysis, and in many other cases the damage to the nerve cells is so slight that in a few months, or a few weeks even, the cells have recovered. On the contrary, some

of the patients with the worst cases may get well with little or no treatment. Those are not children whom orthopedists often see. Many intelligent children are attacked by this disease, and some of them are permanently crippled or disabled. Most of them can be improved by proper, intelligent work. In some cases it means difficult, dangerous surgery, but I believe the results are well worth the effort.

POTT'S PARAPLEGIA. DR. JOHN RIDLON.

The purpose of this paper is to direct attention to certain "dreams" that have been repeated down through the ages until they have become accepted as facts, when they are not facts at all. The cause of the paraplegia that is a complication of tuberculous caries of the spine is believed to be pressure on the spinal cord; but whether from inflammatory thickening of the meninges, tuberculous granulation tissue, tuberculous abscess, or narrowing of the spinal canal from the sharpness of the curvature or angulation, cannot be determined except by laminectomy, and frequently not then. Laminectomy usually relieves the pressure on the cord, but it is usually followed by death. Rest in bed or on a convex gas-pipe frame results in complete recovery in all cases—at least that has been my experience throughout more than forty-eight years. The necessary period of recumbency averages more than six months. One case lasted four years, one seven years, and one ten years. Spinal cords compressed by narrowing of the canal from acute curvature or angulation ultimately become adjusted to the restricted space. Tuberculous granulation tissue and abscesses gradually shrink; edema and inflammation of the meninges gradually disappear under prolonged rest, and the paraplegia disappears. Laminectomy is unnecessary; it is dangerous and usually results in the death of the patient.

DISCUSSION

DR. E. W. RYERSON: It would be an unfortunate and unusual meeting of the Orthopedic Club if Dr. Ridlon and I did not differ widely on some subject. I know a good deal about Pott's paraplegia and what produces it, because I have performed laminectomies in several cases and have looked inside to see what was producing the trouble. I operated on three patients last year, and presented them at a staff meeting at St. Luke's Hospital. One patient had been under the observation of orthopedists for several years. He was greatly deformed, and could not be put on a gas-pipe frame because his deformity was too severe. He was seen by two neurologists, who thought laminectomy advisable. The vertebrae had ankylosed perfectly. When a small piece of bone, which could do no harm, was taken out, the spinal cord was found to be flattened out and overstretched. The patient has improved continuously since the operation and is getting better every day. He is well of the Pott's disease, and the paralysis has almost disappeared. The other two cases in which I did a laminectomy showed definite abscesses in the canal, with granulation tissue.

It may be right to keep a patient in bed for three or four years to see if something will not happen to reduce the pressure on the cord (which is the only cause of Pott's paraplegia), but I would not wish to stay in bed for four years when the pressure could be relieved by a simple operation. I believe that in selected cases of Pott's disease, which have not responded to treatment after a reasonable time, it is our duty to perform a laminectomy and give the patient a chance to get well. This operation need not weaken the spine if small bone splints are placed on both sides of the laminae. The stability of the spine is not diminished in the least, and the disease will have a chance to get well.

DR. CHARLES M. JACOBS: Some years ago, at the Cook County Hospital, I gave much time and thought to the study of Pott's paraplegia. I published my observations, but perhaps like those of Dr. Patrick, they were not seriously taken. There are two factors to be considered in compression of the spinal cord in tuberculous spine disease: (1) pressure from tuberculous granulation tissue, which occurs mostly in children, and (2) pressure from an intraspinal abscess, usually seen in adults. Though other factors have been prominently mentioned, they should not be seriously considered.

In my series of seventy-five adults having Pott's disease, examined and treated at the Cook County Hospital, paraplegia occurred in twenty-four cases that were due to intraspinal abscess, and not one complete recovery was recorded. Laminectomy was done in several cases. Five paraplegic patients were examined post mortem, and in three instances the cord was completely severed from pressure caused by the tuberculous abscess.

I agree with Dr. Ridlon that laminectomy should not be performed on children affected with Pott's paraplegia, because when they are treated by recumbency on a Bradford frame, recovery generally occurs. In Pott's paraplegia in adults, I do not advise laminectomy as it does not relieve the pressure.

DR. JOHN L. PORTER: I understood Dr. Ridlon to say that he had never seen Pott's paraplegia that had been proved due to direct pressure on the cord, and also that he had never seen a case occur after the disease had been cured. A number of years ago I was called to see a man who could not get around. He was about 50 years of age and had a spastic paraplegia. When he got up in the morning he could move about the room easily but after dressing he would have a tingling sensation in the foot and paraplegia; by the time he was ready to go to work he would have to go back to bed because he could not move. He said that he had had disease in the spine but had received no treatment for twenty years or more. He had been doing type setting for twenty years and never had to use a cane. Examination showed that he had had a recurrence of the tuberculosis in the same vertebrae and that it had destroyed the vertebrae so rapidly that his weight had produced an increased kyphosis. There was no way to account for the trouble except by increased angling when weight was placed on it. Paralysis was not present when the man was recumbent in bed; after keeping him in bed for three or four months, I made him a tailor's brace, and he was able to resume work. I am convinced that it was direct pressure, without any abscess, on the cord that produced the paralysis.

DR. MEYER SOLOMON: I wonder whether it may not be that the cases in which spinal block, discovered by the Queckenstedt and other tests, exists, would be the ones in which it would be advisable to perform laminectomy, and that the rest treatment would be indicated in the other type.

DR. S. C. WOLDENBERG: In reviewing the literature I find that these cases are not so frequent as formerly, and I wonder if some may not have been the result of malignant growth in the spine instead of Pott's disease. Probably the care given these patients has something to do with the decrease of these cases; therefore I do not advocate operation and think that the rest treatment is just as good.

DR. PETER BASSOE: In children with Pott's disease it is rarely necessary to perform laminectomy, but I cannot agree with Dr. Jacobs that it is not sometimes desirable in adults. In 1913, I saw a young woman who rather suddenly had developed paralysis and anesthesia from the level of the fifth rib down. She was taken to the Presbyterian Hospital and a roentgenogram revealed what

looked like caries of the vertebrae. She was pregnant at the time. It was decided to give her rest in bed and see if it would cure the paralysis and also benefit the pregnancy. In the course of time she had a painless delivery. A month or two later, Dr. Dean Lewis performed a laminectomy, and she made a complete recovery. The paralysis disappeared and she has had several children since.

In regard to the mechanism of the paraplegia, there is another process besides pressure from an abscess and granulation tissue that often causes paralysis in both carcinoma and tuberculosis. The epidural space lying behind the bodies of the vertebrae gives a little room for any process to go on without causing symptoms in the cord. After a time that process, whether tuberculous or neoplastic, will reach the dura and cause adhesions, which in turn may cause sufficient edema or circulatory disorder in the cord to block the motor and sensory impulses. In such cases, a decompressive laminectomy seems to be the rational thing.

I think Dr. Solomon made a good point when he suggested that the Queckenstedt method should be taken advantage of in demonstrating spinal block. We simply make a spinal puncture, use a manometer, and if on jugular compression there is no rise in the mercury we know there is a block. In the cases in which block exists laminectomy should be considered more seriously than in cases in which there is none.

Dr. George W. Hall: Along the line of Dr. Bassoe's remarks about the trouble being located anteriorly and not posteriorly, a tuberculous case demonstrated this well, for while the patient was completely paralyzed in the lower extremities the sensory symptoms were so slight that no level of anesthesia was determined; the kyphosis alone made it possible to judge the location of the trouble. There was an abscess that welled up from the front alongside the cord. Such cases demonstrate that great pressure may be made on the cord with only slight disturbance of sensation.

Dr. Roy Grinker: I have had some cases in which operation was performed at the Cook County Hospital. With the Queckenstedt test and cisternal injection of iodized oil 40 per cent it has been shown that the fluid was blocked at exactly the point where the sensory symptoms demonstrated that there must be pressure. These cases have shown at operation a thick peripachymeningitis encircling the cord, and this could only be removed in its posterior portion. Microscopically, the peripachymeningitis revealed a thick tuberculous granulation tissue. Unfortunately, some of the patients have died. Two of them, however, are alive and are improving gradually. It is true that the operation is not without danger, but in some instances it certainly seems to be indicated, especially in cases in which a spinal block can be demonstrated.

Dr. Patrick: I think a little practical neurology in this discussion may be worth while. By Pott's paraplegia is understood, in the main, a motor paralysis. A person may be completely paraplegic from slight pressure on the cord. That is not a serious paraplegia. If, however, the sensory conduction is greatly interfered with the condition is much more serious. This should always be taken into consideration when a question of interference arises. As long as there is not considerable anesthesia and analgesia, the cord is not greatly involved and there need be no haste about operation. When the sensory symptoms are marked, the cord is seriously compromised and something should be done. The mere presence of spinal block is not significant. Slight compression will arrest iodized oil 40 per cent and will give a positive Queckenstedt test while not dividing any nerve fibers.

I should like to go back for a moment to my statement regarding rheumatism and sciatica. I found on my record cards a diagnosis of arthritis and rheumatism between 500 and 600 times. Of course patients are not sent to me and do not come to me for arthritis. They come or are sent as cases of neuritis or sciatica.

In discussing Dr. Ryerson's paper, when I spoke of the girl who was lying in bed for ten years and said that the result was about the same as if she had been treated all the time, I assumed that the intelligence of all those present would prevent the drawing of false conclusions. I see I was mistaken. I quoted that case not as advocating neglect, but as a caution against drawing hasty conclusions as to therapeutic results following medication, operation, or any other therapeutic procedure.

Dr. Ridlon: I think my paper must be really worth while because I find that all of you who have spoken are still dreaming dreams and not thinking of facts. Dr. Ryerson spoke of three cases in which he performed laminectomy, and says one patient is getting better. I think he is just dreaming that he is going to get better. Dr. Jacobs mentioned the granulation tissue. All of you know that if a patient is kept at rest long enough, on his back, the granulation tissue will disappear, and that an abscess will dry up and disappear if he is left at rest long enough. Dr. Porter told of the recurrence in a case in which a cure was effected. Every one has seen such cases, and he did not know whether the paralysis was due to the curvature or to something else. I said that the curvature did not cause the paralysis, and that the patient could get well of the paralysis no matter how great the curvature was, without operation. That is definite.

I know nothing about spinal block. In my cases of Pott's paraplegia I have seen none with anesthesia high up. If I had such a patient I would send him to Patrick or Bassoe. One case was mentioned in which the cord was flattened. I told you that the girl who was paralyzed for four years and got entirely well also had a flattened cord.

I am trying to get you not to dream about these things, and not to think that you have an effect when you only have a cause. With complete compression of the cord, any one is justified in killing his patient by laminectomy. I think it is a humane thing to do.

Peripheral Nerve Injuries. Dr. Lewis J. Pollock.

Arthritis of the Spine. Dr. John L. Porter.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 28, 1927

William G. Spiller, M.D., *President, in the Chair*

Posterolateral Sclerosis Following Epidemic Encephalitis. Dr. James B. Mason.

The patient was admitted to the Presbyterian Hospital with a history of having had symptoms in 1920 that resembled those of influenza or the onset of epidemic encephalitis. He recovered from the acute symptoms, but in June, 1925, developed diplopia. This did not last more than a few days. Soon after

this, he gradually became incoordinate in all four limbs and developed a scotoma in the right eye. He had much difficulty in buttoning his clothing, and complained of paresthesias in all four limbs. Since then the upper limbs seem to have recovered almost entirely; yet he still has some paresthesias in each hand. The biceps and triceps reflexes are slightly increased but equal. The recognition of posture and stereognostic perception are normal in each hand, and all cutaneous sensations are normal. In the lower limbs incoordination is marked; walking is difficult without a cane. There is also moderate bilateral weakness of the lower limbs, bilateral exaggeration of the tendon reflexes, bilateral ankle clonus, bilateral Babinski sign, bilateral impairment of recognition of posture in the great toes and bilateral impairment of vibratory sense below the knees. The laboratory observations are all normal.

DISCUSSION

DR. W. B. CADWALADER: The patient has all the symptoms of posterolateral sclerosis of the spinal cord, which developed after epidemic encephalitis. Implication of the spinal cord in epidemic encephalitis is relatively uncommon; yet it is known to occur. Both Dr. Winkelman and Dr. Spiller have reported cases, with necropsy, in which lesions were found in the spinal cord in association with lesions in the brain. Epidemic encephalitis may implicate the central portion of the medulla oblongata and produce symptoms that resemble those of posterolateral sclerosis of the spinal cord by affecting the lemniscus and the pyramidal tracts. I reported cases before this society a year ago, in which I thought this was a possibility. (Lemniscus Symptoms Following Epidemic Encephalitis, *Arch. Neurol. & Psychiat.* **16**:605 [Nov.] 1926).

ATYPICAL NEURALGIA. DR. TEMPLE FAY.

The three cases presented are the first of their kind on record. They have definitely established certain factors regarding atypical neuralgia, other than true trigeminal neuralgia. They establish the functions of the seventh, ninth and tenth nerves in their intracranial component. The great intermingling of these structures with adjacent nerves after leaving the cranial fossa has left the determination of their nuclear and root functions individually a matter of conjecture. By sectioning these nerves close to their point of origin along the lateral aspect of the medulla, before they have had the opportunity to give off branches to one another, observations of their individual function have been possible.

CASE 1.—*Intracranial section of the ninth nerve.*

This patient (E. B.) suffered from carcinoma of the base of the tongue with metastasis to the anterior cervical lymphatics. Pain in the tongue, lower jaw, throat, ear and back of the neck, radiating to the vertex, was the reason for seeking relief by section of the posterior roots of the following nerves: (1) glossopharyngeal, intracranially; (2) second and third cervical; (3) trigeminus.

The following observations were presented: After section there was complete anesthesia of the face, neck and head, with the exception of a small zone in the posterior concha of the ear below the pinna and just behind the ear This zone therefore must be attributable to either the tenth or seventh nerve. There was no cutaneous area attributable to the ninth nerve that could be detected either in the region of the ear, behind the ear or in the throat. There was no loss of the gag reflex. Weakness of the soft palate could not be demonstrated. Pain function apparently remained in the posterior pharynx.

There was loss of the sense of taste on the left side. The case is unique in that not only was the ninth nerve sectioned intracranially to determine its value in relief of pain in the throat and ear, but the adjacent sensory fields were also destroyed by section of the fifth nerve and the cervicals.

The patient was relieved of all pain except the pain referred to the ear and throat, so-called glossopharyngeal neuralgia. It is therefore a matter of conjecture as to whether the ninth nerve in its intracranial portion has a sensory value other than that for the special sense of taste, as the ninth nerve apparently does not carry the fibers which supply the cutaneous areas about the ear or fibers which supply the posterior pharynx with pain.

CASE 2.—*Intracranial section of the vagus.*

This patient (J. C.) likewise suffered from carcinoma of the base of the tongue with metastasis to the anterior cervical lymph glands. He complained of pain in the lower jaw and tongue and pain deep in the ear and radiating down the throat. In view of the failure to relieve the pain by section of the ninth nerve in the former case, the posterior root of the fifth nerve was destroyed in the portion supplying the lower jaw, and the second division. This relieved the pain in the tongue and the lower jaw. It had no influence on the pain deep in the throat or ear. Subsequent section of the vagus nerve on the left side was undertaken by the intracranial route, and the fibers were destroyed. Following section of the vagus, pain was relieved. Anesthesia was produced in the posterior aspect of the concha, the ear, the pinna and just below and behind the ear. This was the exact zone which had remained intact following section of the ninth, fifth and cervical nerves in case 1. It was obliterated by section of the tenth nerve.

It is therefore evident that this area of the ear is supplied through the tenth nerve, the ramus auricularis nervus vagi. Furthermore, it would appear that the pain in the throat and ear, as evidenced by these two patients was of vagus origin, probably through the superior laryngeal nerve, referred to these cutaneous zones of distribution of the ramus auricularis nervus vagi.

In this case there was paralysis of the vocal cords on the left and paralysis of the soft palate, loss of the gag reflex and paralysis of swallowing. Attention is called to the fact that the zone of anesthesia produced by section of the vagus is identical with the zone of zoster manifestation pointed out by Hunt, and that this zone, being a factor of the tenth nerve, manifested itself probably in Hunt's cases by spread of inflammation from the geniculate ganglion to the ramus auricularis nervus vagi, which often fuses with the seventh and lies close by. The finding therefore would seem to bear out the fact that the seventh nerve does not contain fibers of common sensation to this portion of the ear. Further attention is called to the fact that the actual motor supply for the vocal cord lies in the tenth nerve, in its intracranial root, and not in the vagus accessory as is claimed by some. The gag reflex is a manifestation of the tenth nerve. Innervation of the soft palate comes through the tenth. Pain referred to the throat and ear, the so-called glossopharyngeal neuralgia, is in reality a function of the tenth nerve, as this patient was completely relieved of pain after section of the vagus, the pain being similar to that in the former case which did not yield to section of the glossopharyngeus.

CASE 3.—*The final result of section of the posterior root of the fifth nerve, the resection of Meckel's ganglion, stripping the common carotid, ablation of the superior cervical ganglion and trunk. Section of portion of the seventh nerve.*

This case is of extreme interest in that the patient showed the so-called "Sluder syndrome" of pain below the eye, traced along the zygoma back toward

the ear. The sphenopalatine ganglion was removed and the posterior root of the fifth nerve was cut without affecting the pain. The common carotid artery was stripped with partial relief of pain. The superior cervical ganglion was removed, with complete relief of pain. During operation on the sphenopalatine ganglion, the upper branch of the seventh nerve was sectioned (transzygomatic approach). In view of the anesthesia produced by destruction of the posterior root of the fifth, this gave an ideal opportunity for comparing the two zones, that of the forehead with that of the cheek, the forehead being without either the supply of the fifth or seventh nerve, the cheek being without the supply of the fifth nerve but still retaining that of the seventh nerve.

The case is unique in that it offers opportunity for study of two adjacent zones for deep pressure sense. On careful testing it was found that deep pressure sense was lost over the forehead when the seventh and fifth nerve supply had been destroyed. It was present below the eyes when the seventh nerve supply still remained, even though the fifth nerve supply had been destroyed, all other forms of sensation being absent. It would therefore seem evident that the seventh nerve does carry deep pressure sense as was pointed out by Davis in his experiments on the cat, and first called attention to by Spiller in 1906. The so-called geniculate zone of Ramsey Hunt in and about the ear, on the posterior aspect of the concha, in reality shows that this zone is supplied by the tenth, and not the seventh nerve, the inflammatory lesions being manifested by involvement of the ramus auricularis nervi vagi, which often fuses with the seventh nerve before leaving the stylomastoid foramen and is distributed with its fibers peripherally.

This case further represents the fact that the so-called "Sluder syndrome" may be due in some instances to disease of Meckel's ganglion, but in other instances it is probably due to a disease much lower placed in either the thorax or abdomen with referred pain by means of the higher sympathetic arcs to the face. This will explain the reason that other observers have not been able to confirm Sluder's observations in many cases, though it is well recognized that certain cases do yield to sphenopalatine injection or destruction.

Three of my patients who have been under treatment for atypical neuralgia of the right side for a period of from three to four years, with some relief of symptoms due to injection of the sphenopalatine, subsequently developed symptoms referable to the gallbladder and were operated on for this condition. After removal of gallstones in three patients, the atypical neuralgia cleared up spontaneously. In another case, I discovered a lesion in the apex of the lung —the pain in all four of these cases being referred to the face as in the true "Sluder syndrome." This may be a coincidence, but I suspect not.

It therefore seems evident that pathologic changes in the chest or abdomen may refer to the higher arcs evidences of irritation which are relayed forward into the face. Destruction of the sphenopalatine ganglion therefore in these cases will not relieve pain, as the pain is similar to that seen in amputation neuromas, where, even after the member has been removed, the pain is often referred into the extremities which have long since been absent. A case of a patient operated on this week seems definitely to establish the fact that the superior cervical ganglion acts as a higher relay arc station in the transmitting of this pain to the face. The patient operated on by Dr. Elsberg with section of the posterior root of the fifth nerve for pain of the atypical type, was later operated on by Dr. Stookey, with section of the jugular and carotid branches just above the superior cervical ganglion. The patient still had pain in the face and was extremely tender to pressure over the cervical sympathetic nerve on the

left side. Removal of the superior cervical ganglion and its chain has produced complete relief of pain in the face on that side.

As in other cases in this series, pain is frequently relieved from reference to the face by removal of the superior cervical ganglion chain, but pressure over the sympathetics below the site of operation produces tenderness, and there is pain sometimes referred to the arm and shoulders, showing that the pathologic change lies below, but does not find a means of expression in the higher arcs after ablation of the superior cervical ganglion. This case is therefore an example of the fact that in some cases atypical facial neuralgia may be due to involvement of the sphenopalatine ganglion, but in other cases, it is probably due to pathologic changes lying deep in the abdomen or chest, and therefore a search for this source of involvement does not lie in the head or neck; it is futile to operate on these patients in the sphenopalatine region. A means of differential diagnosis in this type of case is offered. It will be found, however, on carefully testing the patient, that this pain is most intense when the sympathetic chain is involved by pressure. It therefore becomes a diagnostic sign which I have termed "carotidynia"; I have found it present in all cases of involvement of the sympathetic system, from lesions other than that of true "Sluder syndrome." If patients with cases of this character are not operated on for relief from pain in Meckel's ganglion, but are considered from the standpoint of deep pathologic changes of the chest or abdomen, it will offer less confusion as to observations and results. This evidence of "carotidynia" has not been found present in true trigeminal neuralgia. It is only slightly present in the cases of true "Sluder syndrome" (lower half headache of true sphenopalatine origin). It is always present in those cases of atypical neuralgia in which the pathologic process is thought to lie in the chest or abdomen, with pain referred to the higher arcs through the superior cervical ganglion.

During the acute stage of sinus disease, I have seen the "carotidynia" marked on the side of the atypical neuralgia, and pressure over the superior cervical ganglion referred the pain directly into the face, below the eye, deep in the region of the zygoma. Irritation through the higher sphenopalatine ganglion may therefore produce hyperirritability of the superior cervical ganglion, indicating that this structure is a relay center for arcs both from above and below.

The important feature in the two cases cited is that the pain fibers must enter the cervical cord to reach consciousness, and that these do enter below the superior cervical ganglion whether referred from Meckel's above or the thoracic trunk below. That the superior cervical ganglion does play an important rôle in their transmission (cutting the fibers above the ganglion does not relieve the pain) is due either to pathologic change below or to involvement of the ganglion itself (two ganglions now being studied). Wherever the cause, if tractable and unyielding to local measures, the relief of pain can be obtained only by section of the fibers that carry the sensation to the brain. These fibers enter the cervical cord. Section of the connections of the sympathetic chain with the cord will relieve the pain by interruption of the pain arc to conscious levels; it does not cure the underlying cause any more than section of the posterior root of the fifth nerve "cures" true trigeminal neuralgia. It simply destroys the pathways for the reception of pain to consciousness.

The real cause of trigeminal neuralgia is not known any more than the cause of "atypical facial neuralgia." The relief of pain in both cases lies in destruction of the pain arc. Ramisectomy therefore may supplant resection of

the cervical chain and should be considered in order to avoid, if possible, the resultant myosis and enophthalmos that accompanies cervical sympathetic ablation.

DISCUSSION

Dr. Francis Grant: There is no question about the importance of this work, particularly with regard to its application in the relief of pain in and about the face. I suggested some time ago that block of the trigeminal nerve, either by injection of alcohol into its individual branches or by avulsion of the sensory root, be performed for relief of pain produced by a malignant growth in the face, cheek or tongue. Dr. Fay then took up this work and expanded it. By means of section of the ninth and tenth nerves he relieved pain in the throat and ear, and by cervical rhizotomy pain in the neck which was not affected by operative procedures directed against the trigeminal nerve.

But with regard to the efficiency of cervical sympathectomy and stripping the sympathetic plexus from the carotid trunk in the relief of pain in atypical trigeminal neuralgia, I would take issue with Dr. Fay. I have been through the records of Dr. Frazier's cases diagnosed as atypical trigeminal neuralgia—patients having pain referred roughly to the trigeminal distribution but whose pain is not at all comparable to that of major trigeminal neuralgia. There were eleven in all that had been subjected to cervical sympathectomy or carotid stripping—one or both—in an attempt to relieve the distress. I use the word distress advisedly because these patients have a "burning feeling," a "sense of pressure," a "disagreeable pins and needles sensation," rather than any lancinating, tearing, paroxysmal pain such as typifies the major neuralgia so clearly. Of these eleven patients only one was permanently relieved by interference with the sympathetic supply, in this instance by carotid stripping.

I remember this case well. The patient had burning pain in his lower jaw following avulsion of the sensory root of the trigeminal for what appeared to be typical major neuralgia. Several months after operation he returned with this new complaint which was entirely different from his former pain. Dr. Fay clearly demonstrated the interesting, and I believe original, observation that pressure over the carotid trunk just above the clavicle on the same side increased the pain. He had advised a carotid stripping which was done with complete and immediate relief. Among the ten remaining cases, however, there were four in which a similar picture was exhibited, following avulsion of the sensory root of the trigeminal; two with definite accession of pain following pressure over the carotid trunk. None of these patients were permanently relieved by carotid stripping, which was combined in two cases with removal of the cervical sympathetic chain from the superior to the middle cervical ganglia. The other six patients had atypical major neuralgia, the nature of which has been outlined. In two, both carotid stripping and cervical sympathectomy were performed; in two, cervical sympathectomy alone, and in one, carotid stripping alone. In none was the condition relieved or benefited for any length of time. Furthermore, in all these cases the cervical sympathetic chain and the carotid sheath after exposure were stimulated by electricity. In no instance did the sufferers note any definite increase of the pain following such stimulation nor could they localize clearly any area in which any new sensation appeared when these sympathetic systems were thus stimulated. This, it would seem to me, is evidence that no pain fibers are to be found in these structures. Five of these patients had transient relief from pain for a week or two, but it never disappeared completely and always returned precisely as it had been before. Enophthalmos, ptosis of the eyelid and a contracted pupil followed cervical

sympathectomy. The development of this well known syndrome was the only positive change noted. I have no explanation for the transient relief of pain which sometimes occurred, but simply mention it because it was recorded. I have abandoned this method of attempting to relieve patients with atypical neuralgia because results have been so disappointing. The patient is simply subjected to an operative procedure by which he is not benefited.

Dr. William G. Spiller: Dr. Fay, in this work, seems to have determined that the sensation of the concha and that of the area just posterior to the ear must be from the vagus and not from the facial nerve. He has added an important contribution to the innervation of the soft palate. The tendency has been to regard the nerve supplying the soft palate as the vagus, and Dr. Fay has made it probable that this view is correct.

From Dr. Fay's work it seems conclusive that it is not the vagus portion of the vago-accessorious, but the vagus itself which supplies the vocal cord, although numerous references may be found in the German literature to the innervation of the vocal cord through the vago-accessorius. Dr. Fay has been able at operation to separate definitely the fibers of the glossopharyngeus, of the vagus, and of the vago-accessorius, as they are distinct from one another at their exit through the dura.

A distinction must be made regarding the function of the facialis in superficial and deep sensation. I reported before this society, in 1906 (*J. Nerv. & Ment. Dis.*, 1906, p. 736), two cases in which the gasserian ganglion had been removed, the pressure sensation being preserved although all other forms of sensation were lost. Later, I gave the notes of these cases to two of my interns, Dr. Ivy and Dr. Johnson, who published a paper on the subject.

Dr. Fay in his work has shown, when he cut completely the sensory root of the trigeminus and also cut the upper branch of the facialis, that when the branch of the facialis was cut, there was no pressure sensation, but when the facialis was preserved, the deep pressure sensation was preserved, although superficial sensation throughout the distribution of the trigeminus was lost. This is convincing evidence of the observation first made by me that deep pressure sensation of the face is conveyed by the facialis.

The theory which Dr. Fay has advanced, namely, that disease of the gallbladder may cause remote symptoms is well supported by the work of Michael Lapinsky.

Dr. Charles K. Mills: I really have little more to say about sensation and the seventh nerve than I said in a paper sixteen years ago. I became convinced that the seventh nerve had in it no sensory elements. If I am not mistaken, Dr. Foster Kennedy of New York and Dr. Brynes of Baltimore and Dr. Kidd of England, among others, have come to the same conclusion. Dr. Fay's report is important, especially his conclusion in regard to the part played by the pneumogastric nerve in the distribution of sensation to the face and head.

Dr. G. M. Byrnes: I shall not attempt to review the past discussions which have taken place in this Society regarding the seventh nerve and its sensory component. Dr. Mills has already expressed my views concerning the anatomy of the facial nerve; and he is correct in referring to me as the Baltimorean who shares his views regarding the zoster zone of the geniculate ganglion. It seems that Dr. Fay has clearly demonstrated that the area attributed by Hunt to the geniculate ganglion is innervated by the auricular branch of the vagus nerve. I have never seen a case of herpes zoster oticus alone or associated with facial palsy in which there was any sensory defect in this region assigned to the geniculate ganglion.

Dr. Fay has intimated that the herpetic eruption in the posterior auricular wall and in the cavum might be due to involvement of the auricular branch of the vagus, but I have been of the opinion for some time that facial paralysis might be due to herpetic inflammation of the geniculate ganglion, and that when the condition is associated with herpetic eruption about the ear, neck or face, the eruption is due not to involvement of the geniculate ganglion, but to a simultaneous and similar lesion in the gasserian or in the ganglion of the vagus. It seems that Dr. Fay has furnished the necessary anatomic evidence which makes this opinion more secure.

Dr. Temple Fay: I have little to say except to the question which Dr. Grant raises. In the series he mentions, the first case was successful, and I can testify that sympathectomy was complete, as I advised. In many of the other operations on these patients which Dr. Grant has called attention to I am sure that the sympathectomy on the carotid artery was incomplete, and frequently the superior cervical chain was not completely isolated or recognized; so it is unfair to assume that the results obtained in this series are comparable with the cases I have mentioned. Extreme care was taken to identify and isolate the sympathetic fibers, of not only the entire carotid coat, but also the superior cervical, and middle central glands and chain, with connections to the cervical roots.

The present condition of the patient is a marked contrast to his condition one year ago, when he walked the floor night after night with pain, and now has found complete relief, only after section of the cervical sympathetic supply. I feel that Dr. Grant has lost the point under consideration. I do not believe that sympathectomy is advisable in all cases of atypical neuralgia. I do not believe that sympathectomy reveals the underlying cause of pain, whatever that may be, either in the area of the sphenopalatine ganglion or the chest or abdomen. The fact remains that the arc of referred pain to the face, of the so-called atypical type of neuralgia, enters the spinal cord to reach consciousness through the cervical roots. Relief of this type of pain, if the underlying pathologic process does not yield, must be undertaken from the standpoint of section of the fibers entering the central nervous system, and as demonstrated in these two cases, these fibers evidently passed into the cervical cord. The operation therefore becomes one, when the necessity arises, for relief of this type of pain in the face, of section of the connections so as to destroy the consciousness of pain; just as section of the posterior root of the fifth nerve relieves the pain of trigeminal neuralgia, so section of the cervical connections in atypical facial neuralgia relieves the pain of this character.

The case is presented to illustrate the pathways of pain in atypical neuralgia, and not as a means of cure for this condition.

Hydrocephalus with Cessation of Persistent Epileptic Convulsions After Puncture of the Lateral Ventricles. Dr. Alfred Gordon.

The pathology of organic epilepsy presents a wide diversity of opinions. It is admitted by the majority of observers, however, that the reception of stimuli and the discharge of motor power, namely, muscular spasms in epilepsy, are the province of the motor cells of the cortex. Therein lies the main station to which flow the abnormal nerve currents. As to the original sources of stimulation or irritation, the accumulated anatomoclinical, physiologic and experimental data prevent a large field of localizations in the brain between its anterior and posterior poles, and the site of the lesion may not necessarily be in the motor area itself. The following case seems to be instructive from this

particular standpoint as it indicates the special seat of the lesion and consequently leads to a logical surgical intervention. The case is also important from a therapeutic point of view as it leads to a line of conduct worth while repeating since the results were extremely satisfactory. Although there are no postmortem features in this case, nevertheless the clinical and postoperative data are so striking that the case deserves a place among many other analogous records.

R. E., a boy, now over 4 years of age, at the age of 2, when first seen by the writer, presented a large head with a protruding forehead. He was totally helpless; he could neither stand nor walk. When seated, the trunk would fall forward. He salivated considerably; food would run out of the mouth. He had to be fed with small amounts of liquids. The sphincters were uncontrollable. The intelligence was that of a low grade idiot. He could not learn anything nor imitate others in spite of all efforts on the part of the parents. At the age of 1 he commenced to have convulsive attacks, at times unilateral and at times bilateral in distribution. As he advanced, the spasms became more and more frequent. He soon developed, in addition to the major attacks, spells of petit mal consisting only of sudden loss of consciousness without muscular contractions. In March, 1926, the convulsive seizures became unusually frequent, about five or eight attacks a day.

Examination at this time showed, in addition to the symptoms described, unequal patellar tendon reflexes, the left increased; the left plantar reflex was in extension; on the right side the knee jerk was much diminished and the test for plantar reflex gave no response. Ankle clonus was absent on both sides. The superficial sensations to all forms were apparently not involved. Examination of the eye showed no gross pathologic change except the presence of full and tortuous veins in the fundi. Roentgen-ray examination showed no abnormalities, and the sella turcica was normal. Urinalysis gave negative results. Blood examination showed: hemoglobin, 80; red cells, 3,350,000; white cells, 6,800; polymorphonuclears, 38; small lymphocytes, 48; large lymphocytes, 5; transitionals, 9. The Wassermann reaction with the blood was negative. The child was pale and undernourished. The frequent convulsive seizures kept him in such a stuporous state that it was difficult to feed him. The family history was unimportant with the exception of three uncompleted pregnancies of the patient's mother. The treatment consisted of small doses of phenobarbital in addition to the usual precautionary measures concerning diet and hygiene. As even large doses of phenobarbital or bromides were not effective, antisyphilitic treatment was instituted in spite of a negative Wassermann reaction, with apparent beneficial results at first. Soon, however, the same intensity and frequency of the attacks returned. Radical measures were then proposed to the parents, which they promptly accepted. In the presence of the hydrocephalus and in view of the anterior fontanel being unclosed in spite of the age (the child was 3 years old) a puncture of the latter was attempted first. There were no untoward results from this procedure, but it had no beneficial effect on the convulsive seizures during the following ten days. Puncture of the posterior horn of the lateral ventricles was then performed on one side and a small trephining about 2 inches (5.08 cm.) back of and 1 inch (2.54 cm.) above the external auditory meatus; the hollow needle was introduced slowly and horizontally until fluid appeared at the outer end; 4 cc. of fluid was removed. A decided improvement followed almost immediately. On the first two days the patient had but one attack a day; on the following days the attacks became less and less frequent, and finally disappeared. On the fourteenth day the attacks again became more frequent, although not so frequent as before the operation. It was then decided

to puncture the lateral ventricle at the same level on the other side. With the same procedure, 4 cc. of cerebrospinal fluid was withdrawn, and at the same time a spinal puncture was performed by which 15 cc. of spinal fluid was allowed to run out. The child made an uneventful recovery. A cytologic examination showed in both seances twelve and twenty-three cells per field, respectively; no organisms were found.

Since the last operation the improvement is noticeable in every respect. In the nine months that have elapsed there has been no return of convulsions, the fontanel has closed; the child has begun to notice objects and persons, he can repeat and say voluntarily words and some sentences; he can stand and walk without assistance. The continuous progress with regard to the convulsive seizures and to the mentality is at present uninterrupted. The child's bodily health is markedly good. The blood picture has become normal.

The report of this case is warranted at least by reason of the unusual physical and mental improvement following the removal of a small quantity of fluid *directly* from the lateral ventricle. The report seems also justifiable on account of the method of approach in the surgical procedure.

The question of pathogenesis of the epilepsy in this particular case is equally important. It is to be presumed that there has been an acute ependymitis or an acute inflammation of the choroid plexuses which, after the relief of the intraventricular pressure, gradually subsided. Of course there is no guarantee against recurrence of the inflammatory state and reaccumulation of fluid with convulsive seizures as a consequence; but nine months of perfect freedom from convulsions against the status epilepticus which existed before the surgical intervention is strongly in favor of the contention that the intraventricular condition was most probably the direct etiologic factor of the convulsive seizures. This case is an additional confirmation of the conclusions expressed by the writer in a previous anatomoclinical contribution presented at the meeting of the American Neurological Association, June 5, 1924 (*J. Nerv. & Ment. Dis.* [Feb.] 1925); namely, that any segment of the brain possesses epileptogenous properties, and that while the mechanism of epileptic convulsions lies fundamentally in the disturbed function of the cortical motor cells, the impulses of the latter are influenced by morbid foci in any region of the intracranial tissue.

Differential Diagnosis Between General Paralysis and Cerebrospinal Syphilis from the Pathologic and Clinical Standpoints. Dr. N. W. Winkelman.

A brief résumé of the important pathologic distinctions between meningovascular syphilis and general paralysis was given with particular emphasis on the reaction of the membranes in the two conditions. In the former there was excessive infiltration of the subarachnoid space with round cells with moderate perivascular infiltration of the vessels in the cortex, especially the deep vessels, while in general paralysis the infiltration of the membranes was usually minimal, and the vessels were intensely collared. It was felt that these facts explained the differences in the clinical pictures and particularly differences in regard to response to treatment; that in cerebrospinal syphilis the infection probably came to the nervous system by way of the cerebrospinal fluid, while in general paralysis the virus probably enters by way of the blood vessels. On this basis one can readily explain the efficacy of treatment by way of the subarachnoid space in cerebrospinal syphilis and the poor results in general paralysis.

Book Reviews

LES NOUVELLES MÉTHODES SUR LES RÉACTIONS COLLOÏDALES DU LIQUIDE CÉPHALO-RACHIDIEN. By EUGÈNE DE THURZÓ. Pp. 182. Maloine: Paris, 1927.

This book, of 182 pages, with a foreword by Professor Guillain, contains the report of the author's work on the comparative methods of study of the spinal fluid in diseases of the nervous system — particularly those of syphilitic origin — and in conditions of non-nervous origin. The methods include the two-colored benzoin reaction, in which a colloidal solution of benzoin is used, with the addition of a combination of light-green (licht-grün) and brilliant fuchsin; also, the colloidal solution of mastic, with or without the addition of dyes and the colloidal solution of paraffin, all of which are compared with the reactions in the colloidal gold solution of Lange. Special emphasis is laid on the difficulty of preparing a standard gold solution and the difficulties that arise therefrom. The mastic solution is considered much easier to prepare and more sensitive in reaction. In making the tests, great care has been taken to determine the sensitivity of the colloidal gold solution to sodium chloride, and further, to control the reactions with a known positive and a known negative spinal fluid.

A study is reported of the reactions of the spinal fluid to determine what, if any, modification follows the treatment of syphilis of the nervous system. Treatment included intraspinal and intravenous injections of arsphenamine; mixed treatment with arsenomercurials; a few instances of intracarotid injection of neoarsphenamine and other methods of inoculation and vaccination. Conclusions point to the fact that treatment causes a moderation of the reactions in colloidal solutions, though there is never an absolutely negative response. The author also finds that the activity of neoarsphenamine is almost 100 times as effective when given intraspinally as when administered intravenously, and the dose should be correspondingly decreased.

The results are added of considerable work on the reactions in colloidal solutions of the spinal fluids in a number of systemic and mental diseases of nonsyphilitic origin. With carefully prepared colloidal solutions, modified reactions were obtained in some of these instances, but they were mainly negative and none could be counted as typical.

Tests of the spinal fluid reactions in colloidal gold, uncolored mastic, and benzoin solutions following artificial pneumo-encephaly (intraspinal insufflation) showed increase in albumin and in the number of cells. The maximum number of cells appears six or eight days after the insufflation; after from ten to twelve days the structure and function return to normal with a decrease in cells, which, however, persist. The colloidal reactions give more or less characteristic meningitic curves as ordinarily seen in meningitic fluid. This is ascribed to increased permeability of the meninges. The curve, after several days, becomes much restricted. Reactions of a similar degree are found in the Wassermann and globulin tests.

The colloidal gold solution of Sicard-Hagenau is recommended for its simplicity and the necessity of only a small amount of fluid. The author found the solution of shellac of Marchionini very sensitive. The technic is simple, and it has proved reliable.

The formulation of a theoretical explanation of the complex electrochemical process involved in the reactions in colloidal solutions used for testing spinal fluids has proved difficult. The idea of the colloidal protective action is generally accepted. The influence of the electrolytes present on the dispersion of the colloid must also be considered. The electrolytes have a tendency in all colloidal solutions to transport their electric charge on the surfaces of the colloidal particles, each cation tends to produce a positive charge, and each anion a negative charge. The hydrogen ion concentration also exercises an influence on the electric charge of the colloid. The chemical reaction of the medium plays a part no less important. It is known that certain albuminoid substances have a positive charge in an acid solution, which becomes negative in its progressive passage to alkalinity. In this process, when the positive and negative charges neutralize each other, the stability of the colloidal solution is least, and the iso-electric point constitutes the optimum for the precipitation. The fact that complete precipitation takes place at the iso-electric point or the point of electric neutrality seems to be important.

The book contains figures showing the curves obtained with the various methods, and with the spinal fluids in different clinical conditions. There are also two interesting charts: one gives the results of the complete serologic study, comparing the different methods, in a series of forty-three cases of widely different diagnoses; the other shows the results of the tests following treatment by different means. Colored plates also show the reactions of the different colloidal solutions graphically.

As a manual for serologic study, the book should prove of interest and usefulness to those working with colloidal reactions.

LES SYNDROMES HÉMIANOPSIQUES DANS LE RAMOLISSEMENT CÉRÉBRAL. By SUZANNE SCHIFF-WERTHEIMER. Price, 22 francs. Pp. 155. Paris: Gaston Doin et Cie., 1926.

Clinical and pathologic studies in cases of well localized vascular diseases of the central nervous system have been successfully made by Foix and his pupils in recent years. In continuation of this work, the author of this monograph collected a large number of reports of cases showing a homonymous bilateral hemianopia clinically. The autopsy in these cases revealed well circumscribed areas of anemic softening of the brain. An attempt was made to correlate the other clinical symptoms with the anatomic observations.

In the first part, a detailed description of the vascular supply of the optic tracts and its cerebral centers is given. Without being given an active part in the discussion, the different theories on the localization of the optical visual center and on the projection of the retina within the external geniculate bodies are recorded. For practical purposes, the following divisions were accepted: optic tract, the region of the corpora quadrigemina, the optic radiation and the cortical centers. The optic tract is supplied by the anterior choroid artery; the region of the corpora quadrigemina, by the anterior and the posterior choroid arteries; the optic radiation, by the middle cerebral artery, and the cortical centers only by the posterior cerebral artery.

The final chapter of the first part deals with the methods of examination and the determination of the gnostic visual fields. Among the large number of hemianopias, not a single case of involvement of the maculae could be detected. In three cases, perception of light in the visual fields was slightly involved. The extent of the hemianopia was always fixed from the onset, and did not

vary during the progress of the disease. The extension of the macular fields was from 10 to 15 degrees and only exceptionally between 5 and 20 degrees. The gnostic vision (perception of objects) corresponded approximately to the fields for blue and red, and was always abolished to the same extent as the perception of colors in the hemianopias.

Chapters IV, V and VI deal with hemianopic syndromes in lesions of the posterior cerebral artery, the sylvian artery and the anterior choroid artery. In reading these chapters one is under the impression that the author tries to simplify the large variations of clinical syndromes that may arise from localized lesions of small isolated branches of the arteries mentioned above. She condenses the clinical syndromes into a few groups, which are anatomically defined by gross lesions of the main branches of those arteries. However, one may concede that this method is of advantage for didactic purposes. The clinical syndrome of the posterior cerebral artery consists of the thalamic syndrome of Dejerine and Roussy and of homonymous hemianopia. In lesions of the left hemisphere, they are combined with aphasia and a more or less pronounced alexia. The syndrome of the sylvian artery is grossly a hemiplegia combined with homonymous hemianopia on the same side, sensory disturbances mostly marked at the extreme parts of the arms and legs, and motor aphasia if the left hemisphere is involved. If only the left superficial or deep branch of the sylvian artery is involved, the intensity of the apraxia may help to localize the lesion. The apraxia is only moderate and transitory in softening following lesions of the superficial branch; it is marked from the beginning in lesions of the second branch. Hemianopia is mostly absent in lesions of the deep branches. In lesions of the superficial branches, hemianopia is produced by subcortical softening or frequently by involvement of the parietotemporal region. The syndrome of the left anterior choroidal artery consists of a right hemiplegia without aphasia, of a right hemianopia and of moderate right hemihypesthesia. If the left artery is involved, a differential diagnosis with the involvement of the right sylvian artery is difficult, despite the character of the hemiplegia.

In five cases of bilateral hemianopia, four presented a persistent and marked ideomotor apraxia. Quadrant hemianopias were noticed in lesions of the posterior cerebral arteries, with a predominating involvement of the upper right or left quadrant, the perception of colors being normal. Cortical blindness was noticed in one case following disseminated sclerosis. Two cases showing moderate involvement of one cuneus clinically did not show hemianopia.

A bibliography of the publications on which this work is based adds to the value of the book. The monograph itself may help the clinician in perfecting the exact localization of cerebral vascular lesions.

FREUDIAN ESSAYS ON RELIGION AND SCIENCE. By CAVENDISH MOXON (M. A. OXFORD). Pp. 133. Price, $2.00. Boston: Richard G. Badger, The Gorham Press, 1926.

It is the avowed object of this small collection of independent essays on the freudian theory to "stimulate readers to further study of the psycho-analytic discoveries." As it has come to be recognized among the acknowledged disciples of Freud that without having undergone a personal analysis a just and wholly unbiased attitude toward the doctrine is impossible, as in fact the author states in his preface, one hesitates to venture among the pitfalls of criticism, especially if one believes that there may be something in their claim. Even

if one admits its partial truth, the patronizing attitude it engenders is altogether unfortunate, as exemplified in the following statement: "Still the fact remains that the not too neurotic unanalyzed reader is able to gain from the careful study of psycho-analytic books some insight which can be used in erotic, social and educational relationships, to avoid the more obvious mistakes and to improve psycho-physical health."

One would, no doubt, be more appreciative of this doctrine if its adherents could somehow be more appreciative of one's capacity to grasp its significance. The usual attitude of aloofness tinged with a certain superiority is not likely to win the doubting to allegiance, even though they may show a willingness to learn and comprehend. In these essays the author does not ingratiate himself with the "unanalyzed" reader, nor is his choice of topics altogether happy if his aim is to instruct. He attacks large problems in no uncertain fashion, and reaches apparently entirely satisfying solutions through the freudian mechanisms, with which, naturally enough, the casual reader finds it difficult always to agree.

There are eleven essays in the volume beginning with "Religion in the Light of Psycho-Analysis" and ending with "Freud's Death Instinct and Rank's Libido Theory." In the intermediate chapters the Christian creed, immortality, epileptic traits in Paul of Tarsus, dreams, criminology, freudian theory and sexual enlightenment, Coué and Nietzsche are treated from the psychanalytic point of view. The discussion is readable, convincing in places, difficult to follow in others, and permeated throughout with the attempt to bring under one category of explanation all activities of the human mind, whether expressed in terms of personality or doctrine of whatsoever character. Few will now question that the ideas introduced by Freud are of wide-reaching significance and great practical importance, but I am inclined to think that their influence would be even greater if there were less effort to force into the freudian categories the entire range of human experience.

One reads such a series of essays with keen interest and respect for the author's mastery of his subject matter, but also with a certain antagonism that can hardly be wholly the result of the fact that one in "unanalyzed" and therefore incapable of fully appreciating the cogency of the reasoning. Perhaps it is too much to demand the same liberality of thought on the part of the freudian which he expects from his critics. It is nevertheless to be hoped that sympathetic interpreters of the psychanalytic doctrine may arise in increasing number to reconcile that doctrine with the mass of human knowledge that it may supplement but cannot replace. Moxon is not one of these. As a partisan exposition of freudian psychology, however, the book may be cordially recommended.

NEUROLOGIE, NEUROPATHOLOGIE, PSYCHOLOGIE, PSYCHIATRIE. Memoires, publiés á l'occasion du jubilé du PROF. G. ROSSOLIMO, 1884-1924. Pp. 981. Moscow: Narkomzdrav- Glavnaouka, 1925.

This "Festschrift" in honor of Professor Rossolimo is written for the most part in Russian, with a sprinkling of French and German papers and one lone contribution in English by William G. Spiller, on "Endotheliomatous Infiltration of the Entire Spinal Pia and of the Pia of the Base of the Brain." The Russian articles are abstracted in French; the English, German and French papers, in Russian. Practically every known subject in neurology and in psychiatry and its allied subjects is covered in this splendid work. There are

in all 104 authors. The subjects are grouped as follows: (1) Social psychoneurology, containing articles on narcotism and criminality, psychology of the masses and others; (2) morphology and physiology, containing "Essay on the Glands of Internal Secretion," "Studies on the Localization of Function of the Frontal Lobes" and so on; (3) psychology and psychotechnic; (4) general pathology and pathologic anatomy containing two contributions that are of great interest and importance, one (in French) by Cecile Vogt, "L'état marbré of the striatum," and the other (in German) by her husband, Professor Oscar Vogt, "A Further Contribution to the Elective Nature of the Patho-Architectonic Changes of the Cerebral Cortex." (5) Under the head of diverse themes on clinical neurology important contributions appear: Professor Lapinsky, on the mechanism of intercostal neuralgia, gives a short account of conditions producing intercostal neuralgia, a subject that has recently been studied intensively by Carnett of Philadelphia. Kron gives a summary of the differential points between multiple sclerosis and syphilis of the nervous system. Marinesco has a new contribution to the study of the microscopic changes of multiple sclerosis. Prof. Seletzky's report of a case of lesion of the epiconus is interesting. (6) Among the papers in the section on tumors of the nervous system are Spiller's; Kojewnikoff's contribution on the diagnosis of tumors of the spinal cord, and one by Filimonoff on study of primary and secondary carcinoma of the central nervous system. (7) Under the head of epidemic encephalitis, Professor Pappenheim has a study of upward spasms of the eyes in postencephalitic parkinsonism, and Tschetverikoff discusses the pathologic anatomy and pathogenesis of postencephalitic Parkinson's disease. (8) Psychoneurology. (9) Under the head of various themes in psychiatry, Marie and Levaditi discuss neurotropism and general paralysis. (10) Under the subject of surgery appears a contribution by Nonne on the question of the diagnostic and therapeutic occipital puncture, and Rosanoff discusses traumatic epilepsy and its treatment. The final grouping consists of four contributions under the heading of therapy, two of which focus attention on ionization by means of the galvanic current in the treatment of facial palsy and tic. The lack of contributions on treatment is a sad commentary on how rich in scientific facts neurology may be and how poor in therapy.

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